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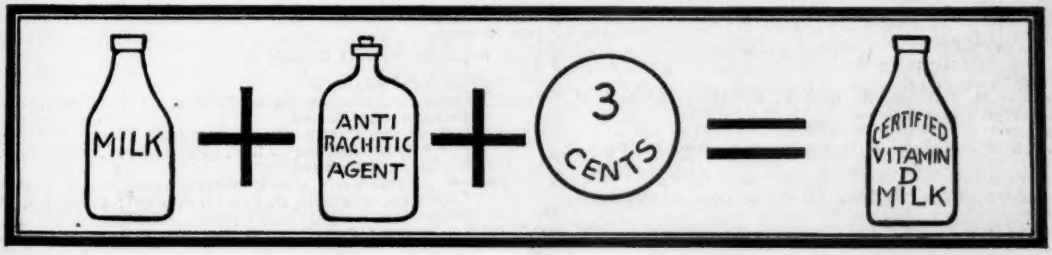

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The next Annual Meeting of the Massachusetts Medical Society will be held in Boston, at the Hotel Statler, June 3, 4, and 5, 1935.

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NEW ENGLAND BRANCH, AMERICAN UROLOGICAL ASSOCIATION

TRAUMATIC RUPTURE OF KIDNEY WITH SYMPTOMATOLOGY OF ACUTE ABDOMEN*

With Case Reports

BY HAROLD S. BACKUS, M.D.†

FOR quite some time I have been interested in retroperitoneal pathology causing as outstanding symptoms those of acute peritonitis, especially injuries to the kidneys with retroperitoneal hemorrhage. Realizing the difficulty in differential diagnosis of acute peritonitis and retroperitoneal hemorrhage, I carefully sought the literature and all textbooks and was surprised that no mention was made of retroperitoneal hemorrhage causing these symptoms except in the *Lancet* in 1921. There Dr. Bonar, of London, gave a short report of two cases.

His first case had symptoms in every detail of early peritonitis (right-sided pain with right rectus rigidity, nausea and vomiting, and high white count). The preoperative diagnosis was a question of acute cholecystitis or abscessed appendix. At operation no intra-abdominal pathology was found, but there was a large retroperitoneal hemorrhage in the region of the right kidney. One year later this kidney had to be removed and a melanotic sarcoma of the kidney was found.

His second case had a laparotomy performed; but the findings were not given. The patient suddenly collapsed and died one week after operation. At autopsy it was found that an abdominal aneurysm had ruptured filling the retroperitoneal space on the right side with blood.

After my first case in 1931, Dr. Cole of St. Louis reported in the *Journal of the American Medical Association* three cases of severe trauma of the back, two with ruptured kidney, and one with impacted fracture of the fourth lumbar vertebra. All had as the outstanding symptoms those of acute peritonitis including elevated white count; all had laparotomies performed and nothing intra-abdominal found; all had retroperitoneal hemorrhage. The two with ruptured kidneys had blood in the urine.

I have two cases to report on traumatic ruptured kidneys with retroperitoneal hemorrhage, giving as the outstanding early symptoms those of acute peritonitis.

CASE 1. An Italian boy, aged thirteen, entered the hospital January 25, 1931 on the General Surgical Service. Three days previously while sliding, he hit a tree with his right side. The next day he vomited and had severe pain and tenderness in the right abdomen. The third day when examined on admission, he presented a board-like abdomen with extensive tenderness of the whole right side. Temperature 102-R. Pulse 100. Respiration 22. First urine was negative. His urine showed blood on the next day. White count 15,400. Neut. 78. Lymphocytes 22. A laparotomy was done in the afternoon. *Surgeon's Report:* The peritoneum appeared normal except in front of the right kidney fossa where it bulged forward and seemed pinkish. A retrocecal appendix was removed. Pathological diagnosis: Chronic Appendicitis. The peritoneum was closed with a drain and reflected forward to explore the right renal fossa. The fatty capsule was found ruptured and free blood present. The kidney was not exposed. A drain was inserted and brought out through a stab wound in the lower back. The main wound was closed in layers around the peritoneal drain. On the fourth day postoperative, sepsis began to develop and a very stormy convalescence began. Pus drained freely from both wounds for weeks. February 7 (two weeks after admission), a G. U. consultation was requested. Cystoscopy revealed indigo carmine dye, given intravenously, appearing from both sides in normal time and in good concentration. The x-ray machine proved to be out of order and no plates could be taken at this time. I did not catheterize the left ureter for fear of infecting the left kidney. The right ureter catheterized easily and pussy urine was collected. A culture showed profuse growth of B. Coli. One month later (March 3) the Surgical Service changed and another surgeon explored the kidney. He found a very thick indurated mass representing the fatty capsule with multiple abscesses and the lower pole of the kidney necrotic. One rectal tube and two large cigarette drains were put in. This drainage persisted for several weeks more. Several blood transfusions were given during these past three months. May 1 he was transferred to the G. U. Service. He gradually improved and was discharged June 17 in good condition, with sinuses closed and only rare W.B.C. in the urine. (Slides shown.)

CASE 2. A small anemic girl, eight years old, was admitted to the hospital September 15, 1933, four hours after falling off a stoop, about eight feet high, striking her right flank on a log. Her chief complaint was pain in R.L.Q., nausea, vomited (once). P.H., unimportant. *Examination:* Her abdomen was board-like with extreme tenderness to the slightest touch. Her B.P. 120/72. W.B.C. 12,700, 88/12.

*Read at a combined meeting of the New York, Philadelphia and New England Branches of the American Urological Association, at New Haven, Conn., April 21, 1934.

†Backus, Harold S.—Urologist, Mt. Sinai Hospital, Hartford, Connecticut. For record and address of author see "This Week's Issue," page 599.

Rectal temperature 100°. Catheterized urine showed gross blood. The rest of the examination was essentially negative. She was placed in bed and kept quiet. The pain gradually subsided in the L. Q., but remained in the upper quadrant and in R.C.V., angle for three weeks. October 19 (one month after injury) a pyelogram with diodrast excretion showed a normal left kidney, the right enlarged, with the upper calyx and pelvis irregularly filled. The lower calyces were not filled, due to the pressure being changed from the hemorrhage within the lower pole. She ran an evening temperature until the middle of November (two months) although her urine was negative for pus but still showed R.B.C. Cystoscopy (November 5) with pyelography shows the calyces and pelvis filling better, in fact they are enlarged as an evidence that the pressure has been relieved. However, there is considerable contraction at the ureteropelvic junction showing a posttraumatic fibrosis. (Slides shown.)

CONCLUSIONS: Two cases were reported and slides shown of ruptured kidneys with retroperitoneal hemorrhage producing as the early outstanding symptoms those of acute peritonitis. There is very little concerning this recorded in the literature. With the exception of my second case, all that are reported had laparotomies performed.

The convalescence is thereby retarded. With these thoughts in mind we may better choose the proper site to explore.

URETERORECTAL ANASTOMOSIS IN A WOMAN WITH A SINGLE KIDNEY BECAUSE OF TUBERCULOSIS OF THE BLADDER*

BY THOMAS N. HEPBURN, M.D.‡

DR. Spillane has just given you the indications and technic for nephro-ureterectomy for tuberculosis of the kidney†. This procedure he and I both feel shortens the morbidity of these sufferers in a group of cases in which the indications are very definite. The case which I propose to present illustrates one of the bad results of a failure to remove a tuberculous ureter. It also gives me an excuse for making some critical observations on ureterorectal anastomosis which I think are needed.

Mrs. A., aged 26, had a left nephrectomy for tuberculosis three years ago, but the lower end of the ureter was left and the bladder symptoms continued to make her life miserable. Finally the bladder contracted to such an extent as you see in this picture, so that it was no reservoir at all. (See fig. 1.) This poor woman had complete incontinence, having to void every few minutes day and night. Various attempts were made to cystoscope her and catheterize the right ureter, but the bladder was so small and the wall so inflamed that the ureteral opening could not be found. The present x-ray picture was obtained by an intravenous pyelogram. We have, therefore, a young adult woman suffering from painful contractions of the bladder every few minutes due to tuberculosis of the bladder with a single right kidney, and a dilated right ureter. She ran no fever, and an x-ray picture of the chest shows the pulmonary process to be static. The presumption in this case was that the single remaining kidney had become tuberculous. Therefore, any surgical procedure could be only palliative.

With this in mind, a right nephrostomy was done under local anesthesia. The urine from this kidney was very pussy, and colon bacilli were abundant. However, after fourteen successive daily urine examinations for tubercle bacilli, none could be found.

As soon as the bladder was relieved from the stimulation of the urine from this kidney, all bladder spasm ceased, and the patient experienced a welcome relief from pain. She could now sleep all night, so that her natural vital forces fighting infection were greatly increased. Her renal function as measured by phthalein output improved. The nephrostomy opening was maintained for three months, which time we gave for the general improvement of the patient and especially for the shrinking of the distended ureter and improvement in the interstitial ureteritis before attempting further surgery. We now had to choose from the following:

(1) Maintaining the nephrostomy opening as permanent.
(2) Letting the nephrostomy close in the hope that the bladder was healed of its tuberculosis as a result of the rest and would distend to a degree sufficient for a comfortable life.
(3) Implantation of the ureter into the rectum.

I decided that the third alternative gave this woman her best chance for the greatest comfort in life which was possible for her.

Three months after the nephrostomy, her ureter was implanted into the rectum. No inlying catheter as suggested by Coffey, or catgut wick as suggested by Mayo, was used. This anastomosis was not allowed to function for a month, the nephrostomy opening being maintained. After this the nephrostomy tube was clamped off and the patient was very sure she passed urine rectally. However, to verify this we injected indigo-carmin into the nephrostomy tube, and the patient was shown to pass the blue urine from her rectum. Thus, having established the fact that the anastomosis was competent, we took the nephrostomy tube out and allowed the lumbar incision to close. It is now a year following this procedure. The young woman has taken on weight; has a good color; can go to the theatre and church, and does not have to get up at night. Also the ordinary sexual relations are restored to normal. For several months following the operation the bladder used to expel some pussy material every two or three weeks, but this has now ceased and presumably nature has been able to clear the ulcerations of the bladder as a result of putting it at rest.

The rectal urination does not differ from other cases in that her bowels move five or six times between getting up in the morning and ten o'clock.

*Read at a combined meeting of the New York, Philadelphia and New England Branches of the American Urological Association, at New Haven, Conn., April 21, 1934.

†Address not submitted for publication.

‡Hepburn, Thomas N. — Urologist, Hartford Hospital. For record and address of author see "This Week's Issue," page 599.

After that she can go any length of time she wants to without rectal irritation.

This case is presented as showing

1. That a kidney should not be condemned as tuberculous without positive proof, and this proof can only be obtained in some cases by a nephrostomy or ureterostomy.
2. That tuberculosis of the bladder can sometimes be greatly benefited by putting the bladder at rest by means of a nephrostomy.

Contracted tuberculous bladder in patient with single kidney. Note the dilated ureter. The patient had total incontinence.

The ureter has been anastomosed into the rectum successfully after preliminary nephrostomy. The patient has now no pain and sleeps all night without voiding.

FIG. 1.

3. That any plastic surgical procedure on the ureter requires a nephrostomy as surely as any plastic operation on the urethra requires a cystostomy.
4. In contemplating a ureterorectal anastomosis with a single kidney, a preliminary nephrostomy is the only safe procedure. It is especially true where we have a severe pyelonephritis and ureteritis. Sufficient time following a nephrostomy must be given for this ureteritis to subside, so that the anastomosis of the ureter into the rectum can be safely done, and a nephrostomy opening must not be allowed to close until one has proved the success of the anastomosis.
5. Any foreign body lying in the ureterorectal anastomosis, whether this be a catgut wick as suggested by Mayo, or a catheter as suggested by Coffey acts as a drain from the infected rectum up into the ureter and kidney. By its wick action and by macerating a ureteral wall already traumatized by manipulation and cutting off half its blood supply, such a foreign body insures an ascending infection.

OBSERVATIONS ON THE EXPERIMENTAL TRANSPLANTATION OF THE URETER*

BY C. H. NEUSWANGER, M.D.†

PRECEDING the introduction of Dr. Coffey's method of transplantation of the ureters in 1909 there were reported 103 experimental cases with a death rate of 86 per cent.

Since 1909 the death rate has dropped abruptly and has varied with different workers from 10 per cent to 23 per cent both in clinical and experimental cases. Most of the deaths previous to 1909 were due to peritonitis, but the introduction of Dr. Coffey's method and the various improvements which he introduced from time to time, largely eliminated this complication.

We still have, however, the annoying compli-

*Read at a combined meeting of the New York, Philadelphia and New England Branches of the American Urological Association, at New Haven, Conn., April 21, 1934.

†Neuswanger, C. H.—Assistant Attending Surgeon, New Haven Hospital and New Haven Dispensary. For record and address of author see "This Week's Issue," page 599.

cation of infection of the ureters and kidneys following transplantation into the bowel and this is quite uniformly reported by various men.

Biever and Mann carried out extensive experimental transplantations by various methods and they reported that "A normal kidney is rarely found regardless of the method used."

In discussing the report of 160 clinical cases from the Mayo Clinic it was stated that "An acute pyelonephritis follows the operation and cannot be avoided."

Dr. Kirwin and Dr. Vermooten, in reporting a series of thirty-eight experimental cases from this clinic, stated that "no matter how careful our technique or what method we used we were not able in a single instance to find at the time of sacrifice a normal kidney".

The object of this report is to add some information, as to the source of this infection and possible means of avoiding it.

During the past five years, Drs. Deming, Kirwin, Vermooten, and I have done 103 experimental transplantations in dogs, and of this number twelve died. Ten of these deaths were due to peritonitis in the cases of rectal transplantation. In a series of sixty-five cases where the ureter was reimplanted into the bladder or where one ureter was implanted into the other ureter we lost only two cases and both of these were due to hemorrhage.

In going over our work it was very evident that the implantation into the bowel was difficult, that the mortality was high, and that the ureters and kidneys always became infected.

The bladder and ureteral implantations, however, were quite the opposite. The procedure was simple, the death rate was less than 3 per cent and in only one case in sixty-five did the kidneys or ureters show gross infection.

In transplanting the ureter into the bladder or into the other ureter we made no use of the scalpel. The ureter was freed for a short distance cut obliquely, and then with a stitch in the tip of the ureter, threaded into a needle corresponding in size to the ureter, the bladder wall was penetrated obliquely and for a short distance under the mucosa. This stitch was then used to anchor the tip of the ureter. In the cases of the ureteral anastomosis the needle was inserted at one point of the ureter and withdrawn about three cm. below and after pulling the ureteral tip into the other ureter the end was fixed with the same stitch.

We observed in both our clinical and experimental cases that ureteral infections and dilatations set in before the catheters were removed, and therefore the infection must have gained entrance through the ureteral wall and not from a regurgitation of intestinal contents.

This conclusion was also supported by the fact that microscopic sections of the ureteral wall showed an infiltration of white cells.

We also observed that the transplanted ureters in the bladder or other ureter did not become infected even in the presence of a cystitis, and where we could demonstrate a regurgitation of infected urine up the ureter immediately after the operation and over a period as long as fourteen months following the operation. This would suggest that the wall of the bowel is a more potent source of infection than the bladder mucosa.

One of our Staff (Vermooten) also demonstrated that, when the Coffey routine was followed, it was possible to demonstrate regurgitation by clamping the bowel above and below the anastomosis and exerting moderate pressure.

We also observed that our animals recovered more quickly following the bladder implantations than the rectal implantations, in fact many of the rectal cases never completely recovered and throughout their convalescence their septic condition was very apparent.

Although the presence of a valve at the ureteral opening does not prevent infection of the ureter it is still absolutely necessary because direct implantation will result in dilatation of the ureter, and kidney due to retraction of the ureter and a stricture formation. I shall demonstrate this in the slides which I am about to show.

The Coffey method has saved our cases from peritonitis, but we still need a method to save the kidneys and ureters from infection.

(Slides shown.)

From our work it would appear that an implantation of the ureter into the bladder or other ureter can be made a relatively simple procedure and avoids the serious dangers and complications resulting from implantation into the bowel.

It should be possible to use this method in such cases as carcinoma about one ureteral opening, and severed ureter and stricture of the ureter.

SUMMARY

Infection of the ureters and kidneys invariably follows transplantation of the ureters into the bowel and this infection travels by way of the ureteral wall.

Infection of the ureters or kidneys rarely follows reimplantation of the ureter into bladder or other ureter.

Valve formation at the ureteral opening is necessary and helps prevent peritonitis, but has little to do with the resultant infection of the ureter or kidney.

Reimplantation of the ureter into the bladder or other ureter by our present method is more productive of good results than bowel implantations.

DISCUSSION

DR. CLYDE L. DEMING, New Haven: I think I expressed my feeling this morning about ureteral operations, especially where they have to be transplanted into the bowel, or reimplanted into the bladder. In either method we fight infection, and that is a difficult thing to do. We have had, in the last two or three years, several instances where tumors of the bladder occupied the areas of one of the ureteral orifices. We have disposed of that ureter by ligating it as high as we can, opposite the iliac vessels. We have not had any untoward results in about seven or eight cases. There has not been any pain and the patient has not known the difference. We have asked ourselves, "Is there not some way in which we can conserve that kidney for the individual?" Dr. Neuswanger has been working on uretero-ureteral anastomosis, and I feel quite sure that where the tumor of the bladder has not passed through the wall of the bladder and where the ureteral opening must be sacrificed leaving nearly the whole ureter, we could dispose of that ureter by transplanting it easily into the other ureter, and thereby advance our renal surgery in a conservative way.

DR. THOMAS N. HEPBURN, Hartford: I am sorry that Dr. Neuswanger's fine work has not, so far as I gathered, been tried in a human being. I speak because of the difference in the size of the pelvis, that it would be, perhaps, more difficult in a human being than it would be in dogs. I simply wish to rise, now, and express my appreciation of the excellent work that has been done.

DR. O. S. LOWSLEY, New York: *Chairman and Gentlemen*—I merely rise to express my great admiration for the splendid work that has been done here in this clinic. I regret very much, and I think we are wrong in discussing such admirable work. I am enjoying a feast, and I hope that the discussers don't interfere with it too much.

URINARY SYMPTOMS IN RELATION TO TRICHOMONAS VAGINALIS INFESTATION*

BY EDWARD J. MANWELL, M.D.† (By Invitation)

RECENTLY we have had occasion to examine a number of women who complained of frequent, painful or burning urination and who showed on examination little to account for their symptoms other than a profuse irritating vaginal discharge in which the Trichomonas vaginalis was found. It seemed of interest to attempt the correlation of this finding with the urinary symptoms.

The first description of the Trichomonas vaginalis was by Donné in 1836[1]. His description of a round or pear-shaped organism a little larger than a leucocyte, with several flagella at the anterior end and a variable tail was sufficiently accurate to stand, little changed, during the next one hundred years. Since then the flagella have been counted more accurately and the organism more certainly differentiated from its close relative, the Trichomonas hominis, found in the intestine and the Trichomonas buccalis occurring in the mouth. Donné found the organism in vaginal secretions and doubted its pathogenicity. Throughout the remainder of the nineteenth century occasional reports appeared describing this interesting little parasite and speculating on its rôle in inflammations of the genital and urinary tracts. These are well reviewed by Dock[2] in a paper of 1896 in which he also described a case of persistent Trichomonas infestation in a young male. During the past five years interest in the Trichomonas has again revived and a great deal of literature has appeared on the subject, chiefly in regard to vaginal infestation.

A fairly distinct clinical entity has evolved from these reports, the Trichomonas Vaginitis of Greenhill[8] or the Vulvovaginitis Pruritic Trichomonalis of Sayer[11]. Typically these patients complain of an irritating vaginal discharge and vulval pruritus. On examination the vulval mucosa is found to be red and excoriated and the vaginal mucosa bathed in a thin foamy exudate and spotted with tiny petechiae. Examination of this exudate usually shows numerous pus cells and varying numbers of active trichomonads. It should be borne in mind, however, that the organism will be found in occasional females with no associated symptoms other than a mild leukorrhea. A variety of treatments has been instituted, all with some success but none with anything like specific curative results. It has been maintained by some, notably Hibbert[5] and Hesseltine[4], that the trichomonas was not the proved etiological agent and the symptom complex was more probably due to an abnormal vaginal flora in which various strains of streptococci predominated. This view is held to be unjustified by others, notably Stein and Cope[10].

Sayer[11] maintained that treatment of the disease should also be directed to the bladder and thought that the residual habitat of the trichomonas is in the lower urinary tract. He claimed excellent results in a series of a hundred cases in which he merely lavaged the bladder and followed this with frequent instillations of neosilvol. Lewis and Carroll[7] described a female with urinary tract infestation and believed the trichomonas to be pathogenic when found there.

It is not, however, the purpose of this paper to discuss the rôle of the Trichomonas in the production of the recognized clinical entity, Trichomonas vaginitis. It seemed to us that the

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urinary symptoms occasionally described in these cases merited more careful study both as to their actual incidence and as to their associated lesions.

With the help of the Women's Clinic of the New Haven Dispensary it has been possible to obtain the records of thirty-five patients who have been found to harbor the Trichomonas. Thirty-three of these patients were female. Of this group twenty-one had special urological investigation.

The thirty-three females are best considered first. Twenty of these cases were found to have abnormal frequency of urination, nocturia, or voiding oftener than every two hours during the day being considered abnormal. Nocturia was complained of in sixteen cases and diuria in eleven. Some type of dysuria, usually burning on urination, was the complaint in fifteen cases and the combination of frequency and dysuria was complained of in thirteen. Nine patients denied urinary symptoms and in four cases no statement was obtained.

In nineteen cases the urethral calibre was estimated by the passage of bougies or other instruments of known size. Of these nineteen, ten were below 26 F. and were found to be tender and to bleed easily. It should be noted here that the constrictions were always found at the meatus and hence, if one agrees with the observations of Wynne[13], cannot be considered as true strictures. The incidence of urinary symptoms was high, however, in those cases with urethral meati less than 26 F. Seven of the ten had abnormal frequency and six complained of both frequency and dysuria. Catheterized urine from nineteen cases revealed trichomonads in only three instances. This last finding confirms the observation of Stein and Cope[10] who grew the organisms from the catheterized urine of five of sixty-one cases but is not in accord with that of Sayer[11] who stated that it could usually be found and who based his treatment on this supposition.

As yet no conclusions can be drawn from the results of treatment. Many are still being treated and information is not available on a number of others. Suffice it to say that of the thirteen who complained of frequency and dysuria, nine reported relief or improvement. Two of these had merely dilatation of the urethra, lavage of the bladder and instillation of 5 per cent argyrol. Five had vaginal treatment alone by one or more of the standard procedures and two had both urethral and vaginal treatment.

So far we have found the Trichomonas vaginalis in but two males, although Jenkins[6] in an excellent review of the literature found reports of twelve cases and added one of his own.

Our first male patient, a youth of twenty-two, was admitted to the Orthopedic Service complaining of painful joints. He was found to have a polyarticular arthritis. He denied all urinary symptoms. In the course of a search for possible foci of infection, the prostate was examined. It was slightly enlarged, boggy and tender. The secretion contained many clusters of pus cells and numerous motile trichomonads which morphologically resembled those commonly found in the vagina. The urine in the third glass contained many shreds and here, too, motile trichomonads were found. An attempt to culture the organism in ascitic fluid was made but no growth occurred. The prostatic fluid was also plated on blood agar and a pure culture of streptococcus viridans obtained.

The second male patient in whom the Trichomonas vaginalis was found is twenty-six years of age. He contracted a gonorrheal urethritis in 1931 and throughout the next year received adequate treatment. In 1933 following exposure he developed a mild urethritis associated with burning and frequency. The acute phase subsided rapidly and when seen in April, 1934, the patient complained of nothing but a persistent morning drop. Examination revealed a few shreds in the first glass and a slightly indurated prostate. The prostatic fluid contained clusters of pus cells and many trichomonads.

In 1932, May[8] reported the finding of the Trichomonas in the prostatic fluid of a man who previously had a profuse urethral discharge. He believed his case unique. It seems probable that the prostate was the source of the persistent infection described by Dock in 1896[2], Riba[9] likewise antedated the report of May in his article on Trichomonas Urethritis in 1931. He believed the Trichomonas to be much more commonly found in the prostatic fluid than is generally recognized and stated that it had been found in nine of the three thousand smears examined during the three years prior to the report, at the Northwestern University Clinic. Stuhler[12] of the Mayo Clinic reported the finding of the Trichomonas in sixteen of thirty-two thousand prostatic fluids examined. Stein and Cope[10] have recently examined the prostatic secretions of three men whose wives were known to harbor the Trichomonas vaginalis. They found the organism in one only, though one of the others had been treated some time previously for a non-specific urethritis.

SUMMARY

1. Of thirty-three women who harbored the Trichomonas vaginalis, twenty-two were found to have definite urinary symptoms.

2. Seven of the twenty with urinary symptoms had sensitive urethral meati which were smaller than 26 F.

3. Two cases of Trichomonas infestation of the male are described.

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DISCUSSION

DR. G. G. SMITH, Boston: I have had one case of recurring cystitis for which I could find no cause, and I was a great deal puzzled by that. Finally she developed rather more leukorrhea than normal, and though I had examined the vaginal secretion before, I never found any trichomonas, but I then found them. I found them also in the urine, as was the case of some of the men discussed by Dr. Manwell. On treating the urine the bladder cleared up entirely and she has been well ever since. That was some months ago.

COMPARATIVE HISTOLOGY OF THE PROSTATE AND PROSTATIC URETHRA IN MAN, MONKEY AND RAT FROM THE VIEWPOINT OF THE ANATOMICAL ORIGIN OF PROSTATIC HYPERTROPHY*

BY RALPH H. JENKINS, M.D.,† CLYDE L. DEMING, M.D.,† AND GERTRUDE VAN WAGENEN, PH.D.†

THE following studies on the comparative histology of the prostate and the prostatic urethra in man, monkey and rat were prompted by the publication of Lower and Johnston's[2] work on the causes of prostatic hypertrophy. Our aim thus being directed toward the cause of prostatic hypertrophy, it was found necessary at the onset to definitely establish the anatomical origin of prostatic hypertrophy. The next step was to find suitable animals upon which experimental studies could be performed. This preliminary report will deal with the discussion of these two factors.

The various viewpoints concerning the anatomical origin of prostatic hypertrophy were summarized by Marion[3] in 1923 after a thorough review of the literature as follows:

1. Those who contend that the pathologic lateral lobes develop at the expense of the normal lateral lobes.

2. Those who think that the normal lateral lobes never give birth to hypertrophy, but that it originates in the periurethral glands as described by Albarran[1], or, in the pre-spermatic group of glands as described by Tandler and Zuckerkandl[5].

3. Those eclectics who recognize that all the glands, periurethral and prostatic are able to give birth to the adenoma that constitutes hypertrophy.

Marion proceeds to declare the first opinion "untenable", the second "indefensible", and the third "unproven". He accepts the idea of the initial growth arising in the periurethral or mucosal glands and hopes for new researches to establish the parts played by the lateral lobes and other glandular tissues.

It is our opinion that the remarkable work presented by Randall[4] in writing about surgical pathology of prostatic obstruction is the proof that Marion asked for. The substantiation of the ideas of what he classed the "Eclectic School", that is prostatic hypertrophy, may arise from the submucosal glands, pre-spermatic and normal lateral lobes of the prostate gland.

We consider it a privilege to be one of the first to endorse Randall's work and believe that our lantern slides showing the anatomical origins and the various types of prostatic hypertrophy arising from them will be convincing.

Hypertrophy of the prostate in the monkey has never been recorded so far as we know. All the anatomical origins are present except the submucosal glands. Absence of these glands makes hypertrophy less likely to occur, as Randall found this type of prostatic hypertrophy the most common form in his series of cases. The pre-spermatic and lateral lobes are beautifully demonstrated.

The rat has all the anatomical origins necessary for prostatic hypertrophy. The submu-

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cosal glands and pre-spermatic group of glands are well demonstrated. We are presenting a typical case of prostatic hypertrophy arising from the submucosal glands in an old rat which we believe to be the first on record.

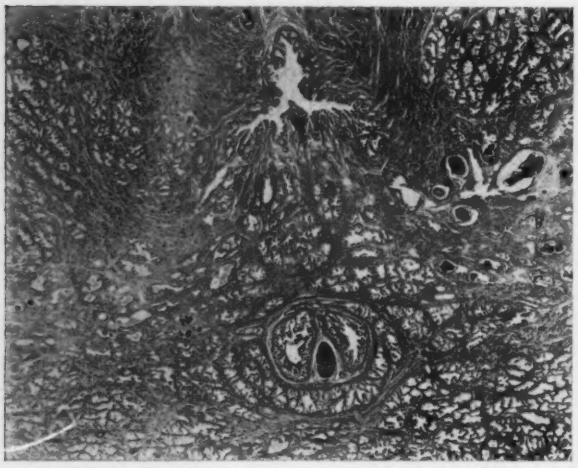

FIGURE 1. The pre-spermatic lobe is situated between the urethra and ejaculatory ducts, extending between and continuous with the right and left lateral lobes of the prostate. This is true prostatic glandular tissue. Two anatomical origins of prostatic hypertrophy are shown here: pre-spermatic and lateral lobes. Surrounding the urethra, the submucosal glands of the prostatic urethra are shown. These glands arise from the mucosa of the prostatic urethra and are not a part of the prostate gland itself. Figure 2 shows these submucosal glands under higher magnification. These glands are another anatomical origin of prostatic hypertrophy.

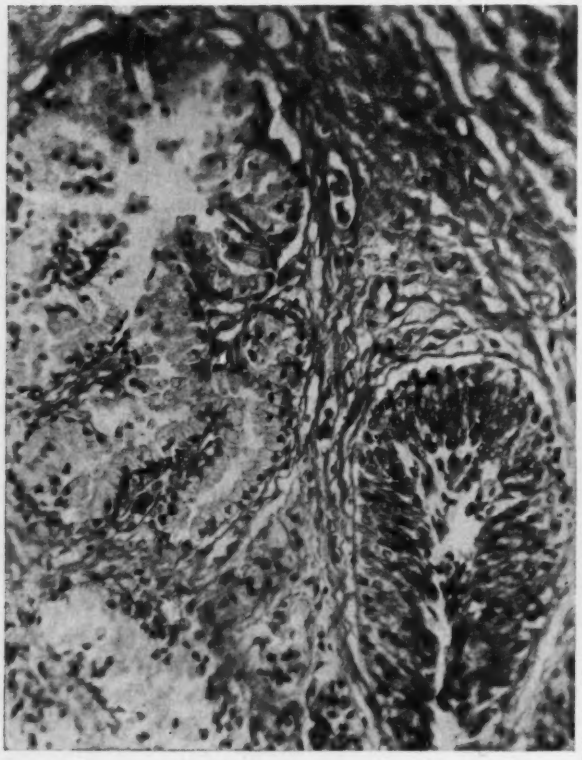

FIGURE 2. The submucosal glands found in prostatic urethra of man 25 years old. The anatomical origin of prostatic hypertrophy of submucosal glands.

FIGURE 3. This is a prostatic hypertrophy that arises from true prostatic tissue. The right and left lateral lobes and pre-spermatic lobe of prostate are hypertrophied. From Randall, 1931.

FIGURE 4. This prostatic hypertrophy arises from the submucosal glands of the prostatic urethra. From Randall, 1931.

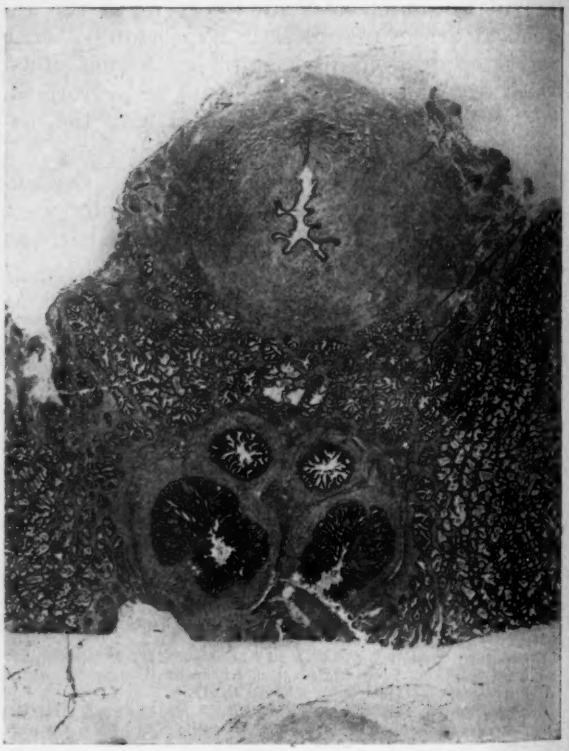

FIGURE 5. The pre-spermatic and lateral lobes of the prostate of a monkey. Cross section through the prostatic urethra and prostate just below the internal sphincter.

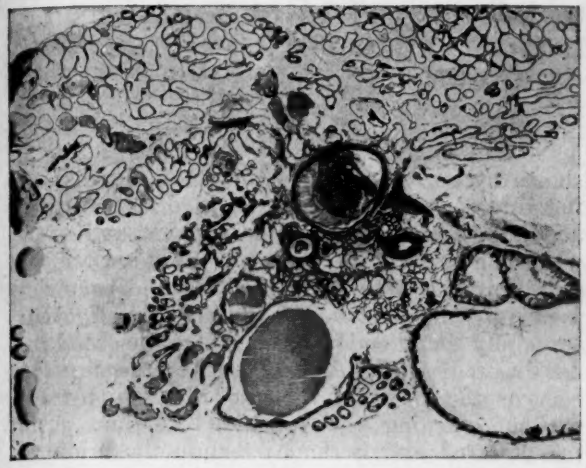

FIGURE 6. The pre-spermatic and lateral lobes of a rat. Cross section through the prostatic urethra and prostate. The pre-spermatic lobe is situated between the urethra and the ejaculatory ducts.

FIGURE 7. The submucosal glands in prostatic urethra of a rat. A cross section through the prostatic urethra just below the internal sphincter.

FIGURE 8. The prostatic hypertrophy of an old rat arising from the submucosal glands of prostatic urethra. The roof of the urethra and bladder opened.

FIGURE 9. This is a cross section of a prostatic hypertrophy of the rat shown in figure 8. It shows the relation of the tumor to the prostatic urethra.

FIGURE 10 This is a cross section of the prostatic hypertrophy of the rat shown in figure 8. It shows the attachment to the urethral wall. The tumor is similar to the submucosal gland tissue.

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THE APPLICATION OF BACTERIOLOGICAL, PHYSIOLOGICAL AND HISTOLOGICAL PRINCIPLES TO PROSTATIC SURGERY*

BY CLYDE L. DEMING, M.D.†

DURING the past three decades there has been so much creditable pioneer work accomplished in establishing the different methods of treating prostatic obstructions that it seems opportune to attempt to correlate the fundamental facts of these methods. Rather than to take issue with the urologists who are advocating these various surgical procedures, we feel indebted to them for their comprehensive reports. Most of them have indicated in their writing in one way or another their respect for certain principles found in the sciences of bacteriology, physiology and histology; but in practical application of related factors, surgery seems to have traveled divergent avenues. Cannot we logically arrange and apply these interrelated basic facts, thereby making our next step of advancement in prostatic surgery?

The bacteriological principles are probably the greatest in number and the most difficult to apply. The opportunities for bacterial invasion vary anatomically as well as with the methods of treatment. The most serious portal is the operative field, the posterior urethra. Of the whole urinary tract it is the area of most rapid absorption of bacteria and drugs. Urethral chills following the passage of urethral instruments, sometimes, even after the greatest precautions against sepsis, are the experiences of all of us. Again, occasionally we see this clinical picture when our prostatic patient voids for the first time following any of the operative procedures. The pre-operative urethral catheter drainage is good in so far as it does not cause much inflammation and allows adequate drainage of the urethra. Successful use of the catheter depends (1) on the first care of the urethra avoiding trauma and infection, (2) on the coöperation of the patient and lastly, on the type of organisms carried into the urethra or the preëxisting organisms in the bladder. Dr. Young's statistics of 98 per cent successful catheter preparatory drainage cases is a compliment to him and to his house staff.

The space of Retzius as a portal of bacterial entry has well been emphasized by Keyes, Bugbee, Kidd and others. Alcock reports in his series of prostatic cases that 70 per cent of the deaths occurred after cystostomy. We, too, have the same experience. It has been definitely shown by autopsy reports that these deaths are not due to sudden release of bladder pressure and anuria; but to infection, sudden and overwhelming in nature. The three-stage suprapubic prostatectomy is based on sound bacteriological truths, but needs further investigation especially in uremic cases. Once the bladder has been drained, it is a logical procedure to remove the prostate through this sinus tract without opening the space of Retzius. The principle of giving the bacterium the minimal opportunity to enter, and after being admitted, to remain as short a time as possible, is a precept not always energetically followed.

The type of the infecting organism is an important consideration. The streptococcus offers serious difficulties from the start. It has been our impression that the streptococcus will be replaced by a second invader of less severity in ten days to two weeks. We never operate on a prostatic case which shows this organism. The hemolytic character of an organism should not be forgotten, for it often presents a formidable barrier to convalescence and increases the mortality. Again, a relatively common organism may become virulent by its own virtue or by the diminished resistance of the host. The ever-changing types of organisms and their character add difficulties to our prostatic surgery. The French have attempted to vaccinate their cases and produce a local immunity prior to operation by injecting vaccine or bacteriophage into the posterior urethra. So far, all writers have indicated and stressed the importance of the posterior urethra and space of Retzius as areas of grave importance respective to infections.

It is quite apparent that none of the surgical operations can be done without a certain amount of concomitant infection in the posterior urethra. We cannot foretell when this will occur, although all clinics are doing a great deal to prevent the frequency of its development. The urethra has an epithelial surface like the skin. Plastic surgeons operating on the face have learned that the best results were obtained by using the scalpel. Since this is true, why should we not expect all operative procedures in the posterior urethra to be done by the use of the knife which will not leave a residuum of sloughing cells? Doctors Caulk and McCarthy have indicated such a fundamental, by attempting to produce an electrocautery which will cut similar to the knife. They are progressing but have not reached our ideal. Thus far, the cautery

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resection leaves not only a cauterized surface but one which is necessarily aggravated by infection. The fact that most of the transurethral cases pass shreds of tissue, mucus, pus and organisms for several weeks suggests infection. Although our patients are relieved of their obstruction and void with ease, but a very few have actually shown a urine free from infection at the end of two months as compared with 50 per cent of the cases operated by the perineal and suprapubic routes. We are not content to believe that a cauterized wound with infection will heal as rapidly as a wound produced by a sharp knife plus infection. Someone should bring forward experimental data with regard to this point.

The physiological type of overgrowth and mechanical obstructions produced should be considered in more detail. It is illogical to remove a large median lobe by the perineal route; for dragging it down through the internal orifice often injures one or both of the sphincters beyond repair. Again, the suprapubic surgeon commits the same mechanical error if he attempts to remove a large prostatic tumor which is wholly extravesical. Kirwin has emphasized the physical and mechanical difficulty in removing transurethrally the large median lobe. Ballenger has attempted to escape this physical barrier by first applying the cautery procedure to the intravesical portion of the enlargement through a suprapubic incision and later resecting the urethral portion transurethrally.

There are several reports which include one hundred or more consecutive cases done by each of the three main techniques. The suprapubic and the perineal surgeons have subjected themselves to certain mechanical disadvantages. The transurethral surgeons have not mentioned the complications of bladder calculi which are found in about 20 per cent of cases, nor the diverticula of the bladder which are found in 8 per cent of prostatic cases. Michel removed fourteen prostatic stones with the transurethral apparatus. So far we have not been so successful, nor do we feel satisfied to disregard bladder calculi and bladder diverticula.

Just where can we apply the transurethral resection method? Certainly, we have found it of particular advantage in the removal of median bars, small median lobes and in the primary bladder neck contractures. For the very fibrous bars and for postoperative vesical neck contractures, the Young punch still has the advantage of all other procedures relative to wound healing and absence of reaction on the part of the patient. After considering the underlying principles of bacterial invasion and wound healing we are hesitant to believe that the transurethral cautery removal of benign intraurethral lesions is at present the logical procedure. On the contrary, for the well-developed carcinoma of the prostate and seminal vesicles with obstruction, the transurethral method is far superior to the other methods. The obstruction is relieved. The patients are comfortable and satisfied.

The third and last point which the author wishes to emphasize is the value of the histological study relative to the various surgical procedures. Since the clinical and histological pictures are often incompatible, we should attempt to remove the whole tumor. Benign lesions may be partially removed by any of the procedures with a complete recovery of the patient. But this is not true of malignant tumors and the only way to cure a prostatic malignancy surgically is by complete removal of the tumor.

During the past year we have encountered two cases of beginning cancer in lateral lobe overgrowth. The cancer is wholly removed as will be seen from the slide. Had a transurethral resection been done there is some doubt in our minds whether the cancerous tissue would have been entirely excised. Vice versa, two other cases who had on rectal examination a large hard nodular prostate with a history of loss of weight and back pains, have clinically, if there ever was such a clinical picture, one of advanced cancer of the prostate and seminal vesicles. Both cases were resected transurethrally, but the tissue removed did not show cancer. Here, the surgical procedure does not give us the tissue we would like to see although we must let our histology dictate to us. Again, may I present tissue from two more cases which had perineal prostatectomy one, eighteen months ago and another, four years ago. The former had a clinical diagnosis of benign lesion and the latter of a malignant lesion. They are histologically undoubtedly cancer, and to-day neither has any urinary disturbance and on digital rectal examination does not show any evidence of local tumor or hardness suggestive of cancer. One cannot say that they are cured, but one could say that with the transurethral method, one would more than likely not remove the cancerous tissue so completely as did the perineal procedure. Suprapubic removal would have been difficult. Radium may, where available, be of value but, unfortunately, we do not all have access to this method of therapy. Because of our experience with such histological findings, our zeal for complete removal of the tumor tissue wherever possible is projected forward.

When we summarize our experiences in prostatic surgery, we are amazed at the multiplicity of fundamental factors involved and that the employment of all of them is almost impossible. However, a more logical application of them may be attained if the surgeon will be more flexible and adapt himself to the use of that particular surgical technique which will best fit the demands of the scientific factors involved.

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THE FUTURE OF PSYCHIATRY IN MEDICAL SCHOOLS*

BY A. WARREN STEARNS, M.D.†

BEFORE discussing more particularly the future of psychiatry in medical education it is perhaps well to review briefly the history of the medical school curriculum. Without going back to a too remote period, medical education in this country prior to the American Revolution was almost entirely carried out by the apprenticeship method. Young men apprenticed themselves to practicing physicians to learn the trade as they would learn any other trade. There were relatively few books and so naturally their education consisted more of learning how to do things than in the accumulation of knowledge. Knowledge was still fragmentary and their teachers oftentimes possessed but a moderate degree of it.

With the beginning of the medical schools about the time of the American Revolution, the subject which was particularly stressed was anatomy. So in the early days, just as Greek and Latin became the indices of one's general education, anatomical knowledge became the index of one's proficiency in medicine. At least one half of the entire body of knowledge which the young doctor was expected to acquire was in the field of anatomy. The other half consisted to some extent of descriptions of disease with very elaborate and varied programs for treatment. In those days there were cures for epilepsy and insanity as well as for many self-limiting diseases.

Gradually other subjects were introduced, but for the most part the curriculum was increased by extensions in medicine and surgery. Then came the ever-increasing mass of required subjects so that the medical course was gradually lengthened to four years.

Within the last generation it has been necessary for the medical school curriculum to absorb the tremendous development of scientific knowledge gained through laboratory investigation. This has been introduced more or less forcibly by pioneers in the various fields. A group of medical scholars has become available for full-time teaching and techniques have been multiplied and elaborated, thus absorbing time from actual clinical experience. So rapid was the increase in these techniques that any fourth-year student could actually confound the average doctor with his knowledge of the chemistry of the blood, urine and other body fluids, or with his formulations concerning the physiology of the heart, digestive system or brain.

Next came the specialties. Innumerable subdivisions of medicine resulted from the specialization of the last generation. More and more, departments of medicine and surgery were broken up into special components. This resulted in a tremendous departmentalizing of the curriculum and of the medical school personnel. Theoretically we have courses of correlation and integration, but practically we are dealing with the ambitions of leaders in the various special fields. Almost every special society has a committee appointed whose function it is to deplore the lack of adequate teaching of their specialty and to conduct a campaign to increase the amount of time given to their precious subject.

The obstetricians deplore the utter neglect of their subject and point out the necessity of more time. Incidentally they say that the students coming to them do not know enough anatomy and physiology so that it is necessary for them to review those portions which have to do with their work. So also the nose and throat specialists call attention to the frequency with which their field is used in medical practice and insist that two weeks in a clinic is not enough to learn the techniques of examination, to say nothing of diseases and their treatment. They also lament the lack of fundamental knowledge of anatomy and of physiology. Likewise the roentgenologists can readily show the increasing importance of their work in practical medicine and deplore the almost complete lack of recognition which their specialty has in the medical school curriculum. Orthopedic surgeons have recently made a survey by which they estimate that twenty per cent of all the cases which a doctor has to treat come within their field. It is insisted that altogether too little time is given to their specialty.

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Lastly come the psychiatrists. With twenty-five years of experience in the missionary field they are well versed in the use of propaganda. Having canvassed the fields of law, industry, religion, education and criminology, and attacked the impotence of judges and legal traditions, the brutality and carelessness of industry and its management, the inadequacy of clergymen and religious customs, the backwardness and blindness of prisons, the futility of educational institutions in general—to enumerate but a few of the modern "Lamentations"—they now focus their efforts on the teaching of psychiatry in medical schools. Elaborate studies have been made of these matters. A barrage of propaganda has been laid down, smacking a good deal of high-powered salesmanship. Their analogies and illustrations have been trite enough, but somehow one builds up a resistance to these efforts. Perfection is not of this world and mistakes will happen in the best regulated of lives. And only the mistakes are mentioned. If medicine is being practiced with an expression of deep insight into human nature, this has not come to the attention of the mental hygienists.

However, having resisted the initial onslaught and preserved the bulk of the medical curriculum in its conventional form, one may now soberly ask a few questions concerning the teaching of psychiatry in medical schools. First, let us assume that everything that has been built up in the past in the name of progressive medical education is not thoroughly bad, but on the whole represents the best that human beings have been able to formulate. It should then be accepted as imperfect and constant attempts should be made to delete that which is bad or futile and to put in improvements from time to time. Then let us assume that medical students should be brought into contact with as many of the facts of medical science as possible within the four-year period, and that in addition it is desirable to familiarize them with generally accepted theories. I think it may be assumed that attempts to inject into them twice as much as they are capable of absorbing in a four-year period are as futile as attempts to put twice as much in the stomach as it will hold. Then also let it be assumed that students should be taught the various techniques of medicine which have been found useful in practice and that they should also learn to do those things which have been found helpful in practice.

It should be observed at this point that medical curricula have been undergoing a revision which shows a trend entirely different from the one just noted. There has been an attempt to get back to fundamentals. The time given to specialties has been reduced and medicine and surgery have been given the right of way. Talk is widespread of the importance of the family doctor, of the weakness of super-specialization, and we are casting back, wondering what it was that our ancestors acquired which we seem to have lost. We are asking whether there is a happy medium between the slot machine on the wall where one punches in certain symptoms, drops in a nickel, turns a crank and gets the answer below; that is, the mechanized technician, on the one hand and careless, rule-of-thumb guesswork from the kindly and friendly doctor on the other hand.

Curricula are being consolidated, specialties are being grouped, at last the citadel of anatomy appears to be falling and one sees a reduction in the hours devoted to this honored and conventional subject. Furthermore, required hours are being cut down, in some cases from six or eight thousand to as low as three thousand for the four-year period. This situation calls for some analysis of the factual basis of psychiatric pretentions. If we give more time to psychiatry to what subject shall we give less time? Is psychiatry a medical specialty? If so it should have a definite place in the medical curriculum and should be well taught. Is it a system of philosophy and an attitude toward life or what-not, as claimed by some of its missionaries? If so, it should have no place in the medical curriculum because of the tremendous crowding of strictly medical subjects.

Let us begin narrowly and take diseases which, whatever they may be called today, have once been called Manic Depressive Insanity, Dementia Praecox, Senile Dementia, Arteriosclerotic Dementia, Feeble-mindedness, Epilepsy, General Paralysis of the Insane, Alcoholic Insanity and the Psychoneuroses.

Let us start with dementia praecox. What facts have we to impart to medical students? What theories have we that are generally agreed upon? What techniques that have been found useful? What can we do that is helpful? I have been struggling with this problem for twenty-five years, eagerly reaching out for every new development which offers promise. What can I say to medical students except, "I do not know the nature of the disease. I am not sure that it is proper to conceive of it as a disease process. I do not understand it. Whether it is a disease of the brain or of the ductless glands or of some other body tissue or fluid, I cannot say. I have no special techniques which are helpful in this disease, neither am I able to do very much about it. I can describe some of the symptoms so that you may be able to recognize this condition in a rough way. I can tell you some of the things that have been done in the institutions to care for these poor afflicted creatures. I can tell you some of the situations in which these individuals are found so that by identifying them you may be more familiar with the great social problems of society." And so one might go on through the gamut, with a little more optimism

in the case of syphilis, alcohol and the psychoneuroses.

Relatively few of the faculty or student body of a medical school have philosophic minds. Most of them tend to be interested in the practical and concrete. Research students are relatively few. Is it to be wondered that neither students nor faculty wish to spend more time on dementia praecox? Is it not obvious why medical administrators are unwilling to turn more and more time over to the exploitation of theories concerning this disease? He who promises more than he can fulfill is always in a difficult situation and after all, we are a part of a great body of scientists; we are not selling hair tonic or breakfast food. The earnest student of psychiatry, if he has a critical mind, must here recognize something of an impassé. He must feel that he is in somewhat the position of a practitioner treating diabetes or pernicious anemia or diphtheria a hundred years ago.

In the year 1758, in a treatise on "Madness" by William Battie, occurs this advertisement:

"Among the many good reasons offered to the Publick, for establishing another Hospital for the reception of Lunaticks, one, and that not the least considerable, was the introducing more Gentlemen of the Faculty to the Study and Practice of one of the most important branches of Physick."

Dr. Battie then goes on to tell how defective knowledge is of lunatics and madness and laments that treatment is being entrusted to "Empiricks" and states that "every Practitioner at his first engaging in the cure of Lunacy has had nothing but his own natural sense and sagacity to trust to; except what he may perchance have heard of Antimonial vomits, strong purges, and Hellebore, as specifically antimaniacal."

We smile as we read the rest of his book on the causes and cure of madness and yet, how much farther advanced are we in the main? While we may claim to have kept pace with the ever-advancing frontier of civilization through our humanitarian efforts in improving the care of the insane and through contributions in the field of criminology, child guidance, industrial hygiene, education, and so on, it seems to me that we must confess a blocking in the really legitimate field of psychiatry; namely, those forms of mental disease which are ordinarily called insanity.

When a great discovery has been made in medicine it has ordinarily not been necessary to organize societies to promulgate its truth. Within a very short time after the announcement of salvarsan, of the liver treatment of pernicious anemia and of the insulin treatment for diabetes, their use was widespread and was being taught in practically every medical school in the world.

I have faith to believe that when psychiatry solves the riddle, or makes an actual contribution toward the solution of dementia praecox, that within a few months practically every medical student in the world will know of this matter whether or not there is a department of psychiatry in his school.

At this point let us consider what may be the minimum requirement in teaching psychiatry in medical schools, for no subject can have more than minimum attention. In the first place, I believe that pre-medical school training is grossly overweighted in the field of mathematical science. Last year in a study of medical school successes, attention was called to the fact that students who did well in mathematics in high school, tended to do well in physics and chemistry in college and that students who did well in mathematics, physics and chemistry in college tended to do well in medical school. This was held up as evidence of the importance of mathematics as a preparation for medical school. To me, the matter seemed otherwise. It seemed to me as if a screen had been set up at the portal of American medical schools excluding all of those excellent personalities and intellects which did not excel in mathematics. One might infer that the selection of the student body of American medical schools was to some extent weighted against the socially-minded.

I wish more time could be given to the teaching of social psychology in pre-medical years. I also wish that a greater clinical point of view could be used in teaching these subjects. Few of the teachers of psychology or sociology have had any clinical or field experience.

It seems to me that under ideal conditions the student, upon entering medical school, should have the personality of the human beings with whom he is to deal brought to his attention by every teacher with whom he comes in contact. As a general proposition it is safe to say that educational attempts should go from the simple to the complex rather than otherwise. Oftentimes we are inclined to deal with the minutiae and complexities of situations rather than the surface indications. I believe that this is reversing the natural order, so that if students could be attracted to this side of their work and have its importance indicated at all points in their education it would be very helpful.

One is astonished in reading hospital records to find the great detail with which a cut or bruise or movement of the bowels is described. Charles Dickens could do no better. And yet oftentimes, the personality of the patient, whether or not mental symptoms are present, is left out entirely.

The task of so sensitizing the student's mind that he is always interested in an appraisal of the personality with which he has to deal, need not be considered that of a medical specialty.

I believe that every teacher in a medical school ought to feel this to be part of his duty. The department of psychiatry should of course furnish leadership in this field.

Again an appraisal of the situation in which the patient is placed and an interpretation of his life history must always remain fundamental. What is everybody's job often turns out to be nobody's job. So it seems to me that every teacher in a medical school ought to give attention to history taking, medical and social. Again the department of psychiatry may well furnish leadership in this field.

The last few years have seen a tremendous diminution in the use of the didactic lecture for the purpose of instruction. Clinical lectures and demonstrations and actual work in the laboratory or clinic have made the lecture seem almost obsolete. For this reason it seems to me unwise to attempt to introduce lecture courses in psychopathology. My experience is that this subject matter bewilders students. I do not believe that it is undergraduate material.

In a general way it may be wise to give a brief series of lectures during the students' first year in medical school simply by way of orientation, this to call their attention to the subject matter of social psychology and medical sociology before their prejudices have been entirely formed on the anatomical side.

During the second year another brief orientation course might well be given stressing particularly the social aspects of psychiatry with a minimum amount of psychopathology.

In the third year the medical specialty should be introduced more specifically. The ideal place to teach this subject is, in my opinion, the neurological or psychiatric department of a general hospital, having the closest relations with the department of medicine, surgery and pediatrics. Students should be brought into contact with the great mass of clinical material easily available in metropolitan out-patient departments. Particular stress should be laid upon attitudes and approaches rather than upon symptoms and diseases. The great mass of psychiatric material coming to an out-patient department presents little promise in medical treatment. The greatest stimulus which can be given the student to interest him in this field comes through the field of social service.

With a properly developed social service department in a psychiatric clinic, tremendous numbers of handicapped, depraved and helpless people can be carried along in a community satisfactorily at minimum cost. If we cannot cure patients with epilepsy and dementia praecox, we can at least make their lot less burdensome both to themselves and their families.

It is particularly important that students learn not only the surface manifestations of emotional disturbances, but the ease with which the deeper layers can be plumbed with a little experience. Personally, I have found it very easy to attract the interest of students in these individuals as social service problems, and very difficult to interest them in abstruse, little understood, and not generally accepted theories of mental disease.

During the fourth year it is possible to attack mental disease as such more directly. At Tufts Medical School we assign every fourth-year student to a state hospital for one month where he serves as a junior interne. We have found this very satisfactory. I believe it is stimulating to the state hospital staff to have students among them. Incidentally they get a lot of chores done which might otherwise prove irksome. But above all, it familiarizes future practitioners with the state hospital service and the problems which they handle. There has been a good deal of isolation in the position of the state hospital staff. This contact tends to break it down. Gradually the practitioners in our state will feel more and more that they are comrades and colleagues of the staffs of the state hospitals.

Lastly, although we have preached mental hygiene for many years and now urge that more training in its subject-matter be given to medical students, I find it very difficult to persuade mature, experienced psychiatrists to spend a morning in a clinic patiently teaching the rudiments of the subject. After all the teacher-student relation has as its most important component something which is neither an administrative device nor pedagogical formulation. The entire activity of psychiatry in the medical school may be quite futile, but the contribution of an experienced, mature man willing to spend his time with students will always be tremendous.

And so our final plan for advance should not necessarily be the seizing of more time from other teachers similarly situated, but should consist of trying each year to do the actual work of teaching students a little better.

TUBERCULOSIS OF THE SPINE IN CHILDREN*

Based on a Study of Eighty-Nine Cases From 1920 to 1930 Inclusive

BY EDWIN FRENCH CAVE, M.D.†

INTRODUCTION: Tuberculosis of the spine is still one of the common conditions treated by orthopedic surgeons. During recent years, however, particularly in this community, this type of case has been segregated to specialized institutional care, and we no longer see it so commonly in our large hospitals. This is fortunate because it is a disease which cannot be treated by short periods of hospitalization, or by ambulatory methods.

The New England Peabody Home for Crippled Children is devoted largely to the care of tuberculous bones and joints, and in this institution the cases are treated under almost ideal conditions. This paper is based upon a study of eighty-nine cases treated at this clinic during the years 1920 to 1930 inclusive. A total of two hundred and sixty-seven patients with all types of deformities was admitted during this eleven-year period; one hundred and eighty-four of these presented tuberculous joints, and of these, eighty-nine were tuberculosis of the spine. During the years 1920 to 1924, there were forty cases of Pott's disease admitted, and from 1925 to 1930, forty-nine cases. The great majority were first seen either at the Children's Hospital of Boston, or at the Massachusetts General Hospital. The average age of admission was 5.3 years. In 1920 to 1924 this average was 5.7 years, whereas, from 1925 to 1930, it was 4.6 years. During the eleven-year period the duration of the disease prior to admission was 22¼ months; in 1920 to 1924 it was 26.5 months, and from 1925 to 1930, 18½ months. This last figure is indeed encouraging, in that it shows that the cases are being hospitalized sooner, and therefore, better results from treatment should be obtained.

TABLE 1

COMPARATIVE DISTRIBUTION OF TUBERCULOUS JOINTS

Total Cases Admitted (Including all types of deformity) 1920 to 1930	267	
Tuberculous Joints	184	68.9%
Tuberculous Spines	89	48.2%
Tuberculous Hips	55	29.8%
Tuberculous Knees	21	22%

ETIOLOGY: Percivall Pott[1] in 1779 stated "The true cause of the disease is a morbid state of the spine, and of some of the parts connected with it; which distempered state of parts will, upon careful inquiry, be always found to have preceded the deformity for some length of time; in infants this is the sole cause, and external violence has nothing to do with it." Pott, of course, was not familiar with the tubercle bacillus, and did not realize that the tuberculous bone lesion is a metastatic manifestation of the disease elsewhere. That the infection is blood borne is hardly without question to-day, and few doubt that the great majority of the infections take place through the gastro-intestinal tract, or the respiratory system.

While acute trauma plays no important part, it is significant that weight-bearing joints are the ones which are most commonly affected. Approximately fifty per cent of the tuberculous joints are represented by Pott's disease; second in frequency is the hip; and third, the knee; (see table 1) so that friction, and the constant wear and tear of daily use are important factors in the etiology of the disease.

PATHOLOGY: Granted that the tubercle bacillus is blood borne, it is reasonable to assume that the lesion in the spine is dependent upon the blood supply to the vertebrae. The intrinsic circulation of the vertebral bodies has been studied by Wagoner and Pendergrass[2]; Doub and Badgley[3] from roentgenological studies concluded that there were three types of tuberculosis of the spine based upon the location of the disease in the vertebrae. They designated the three types as central, intervertebral, and anterior. Of one hundred cases studied they found sixty-one of the central type, forty of the intervertebral, and eight of the anterior type. In our series there were thirty-two of the intervertebral type, thirty-one of the central type, and only one of the anterior type; while eight presented a combination of the intervertebral and central types; two, combinations of the anterior and central types. Fifteen cases we were unable to classify. Doub and Badgley[3] felt that, with the central type, deformities were frequent, whereas, with the intervertebral type relatively little destruction was to be expected. In our experience this has not been entirely true, since we feel that in a number of our cases with the intervertebral type there has been progressive destruction and rather marked deformity although our most marked deformities have come from the central type of lesion. Many of our cases were seen after the disease was far advanced, and it was difficult to tell from the roentgenogram whether it was primarily one

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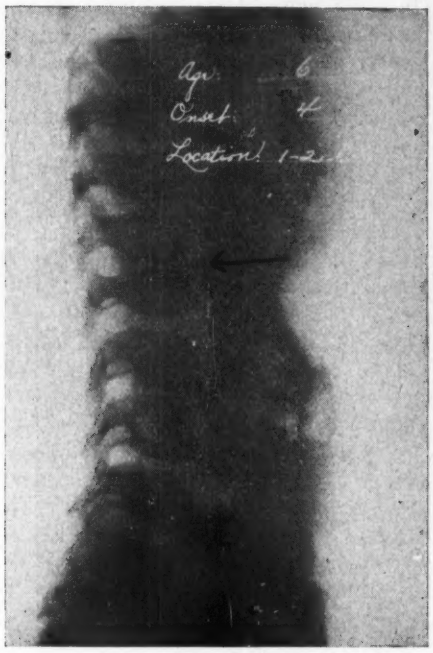
type or the other. We do feel that one very important factor in determining the degree of deformity of the spine in Pott's disease is the amount of destruction that takes place posteriorly. In some cases there had been extensive destruction of the vertebral bodies, but because the disease had remained confined to the bodies, and had not invaded the laminae, pedicles, and posterior supporting structures, deformity was relatively slight. (Fig. 2.)

On the other hand, one case presented involvement of only two vertebral bodies, but had a rapidly increasing deformity, and at operation which was done with the hope of arresting this progress, there was found extensive involvement of the pedicles and destruction of the posterior

(a) Central.



(b) Intervertebral.

(c) Anterior.

FIG. 1. Showing the three types of lesions.

supporting ligaments of the spine. One spinous process was very mobile; this fact is extremely important in determining the speed with which and to what extent the deformity will develop, and it is a very helpful guide as to the prognosis of the deformity. In such cases, consistent conservative treatment will not prevent increasing deformity, and it is in this type of case that more radical measures should be instituted.

FIG. 2. Showing almost total destruction of two vertebral bodies without involvement of the laminae, and pedicles; hence, little gross deformity.

A. S. Bankart[4], in his recent presidential address before the British Orthopedic Association stated, "In all Pott's disease, the natural cures occur only by collapse of the contiguous vertebrae, so that solid bone comes in contact with solid bone, and union can then occur." He stated further, that "It is fortunately hard to prevent collapse of the vertebrae in spinal caries by any means short of operation, and the results of conservative treatment are therefore, generally good, in spite of some misguided efforts to prevent deformity." With this last statement we cannot agree, because it is our belief that every effort should be made to prevent deformity and to insure such correction by operative measures with the hope of preventing collapse of the vertebral bodies one upon the other. Once an efficient intrinsic splint is obtained by operation, it is not necessary to expect apposition of vertebral bodies to insure healing.

LOCATION OF THE LESION: As a rule, the tuberculous disease is localized to one region. In our eighty-nine cases, only five presented double lesions; all of these were in the lower dorsal or lumbar regions. In the eighty-nine cases, there were four cervical, four cervicodorsal, forty-seven dorsal, eighteen dorsolumbar, thirteen lumbar, and three lumbosacral lesions. Undoubtedly, the relative frequency of the dorsal lesions

FIG. 3. Double lesion—5th to 9th dorsal, and 12th dorsal to 1st lumbar.

is accounted for by the increased friction produced by the mechanical situation in this region. It is here also that our greatest deformities occur, due in large measure to the ever-present tendency in this region for one to develop the dorsal curve. Other causes have been advanced as to why this should be true. Ghormley and Bradley[5] felt that pressure from an abscess confined between the anterior common ligament and the vertebral body could destroy bone by pressure, and drew an analogy between this situation and that of pressure on bone from an

TABLE 2

THE OCCURRENCE OF THE ABSCESS CLINICALLY AND BY X-RAY

	Number of Lesions	Abscesses by X-Ray	Abscesses Clinically	Abscesses Present Clinically and by X-Ray
Cervical	4	4	2	2
Cervicodorsal	4	2	1	2
Dorsal	47	31	3	5
Dorsolumbar	18	18	7	5
Lumbar	13	12	10	7
Lumbosacral	3	3	1	1
	89	70	24	22
		78%	26.9%	24.7%

aneurysm. This is undoubtedly true in some cases, but in others the destruction of the anterior portion of the body is due directly to the disease.

SIGNS AND SYMPTOMS: Pain on motion, muscle spasm, and deformity were the most common presenting signs and symptoms of the cases upon admission. Of this number there were eleven pain when moved; of this number, thirty-seven referred pain to the site of the lesion, four had abdominal discomfort, three pain in one extremity, and one suffered from chest discomfort. Thirty-three cases were recorded as having marked muscle spasm, doubtless it was present to a less degree in a greater number. Seventy-one presented some degree of kyphos upon admission. Of this number there were eleven marked deformities, thirty-one moderate, and twenty-four slight. Other less common findings were limp, six cases; night cries, three cases; loss of weight, two cases; difficulty in breathing due to pressure upon the trachea from abscess, three cases; torticollis with cervical lesions, three cases; tenderness over the kyphos, two cases. Of the neurological findings seven cases had definitely exaggerated knee jerks; three were ataxic, two more showed spasticity of the lower extremities, one presented loss of abdominal reflexes, and one absent knee jerks.

DIAGNOSIS: The diagnosis of a tuberculous joint can be established only by biopsy, by culture, or guinea-pig inoculation. In the case of Pott's disease, biopsy is rarely possible. To culture the bacillus is difficult although recent writers, (Blair and Hallman[6]) have been more enthusiastic concerning this procedure. With the lesion which presents a superficial abscess, however, aspiration and subsequent guinea-pig inoculation is a relatively simple procedure. This is rarely possible with the cervical and dorsal lesions, but can frequently be accomplished with lesions at and below the dorsolumbar region.

In the vast majority of cases, the diagnosis can be fairly conclusively made by history, physical examination, and the x-ray plate. With local pain, muscle spasm, limitation of motion, possibly local tenderness and fever with loss of weight, and a destructive lesion demonstrable by x-ray with abscess formation, the clinical diagnosis is tuberculosis. The presence of an abscess, particularly one which contains calcium makes the diagnosis almost certain. A positive tuberculin of 1:1000 dilution or more does not necessarily mean that the particular lesion under observation is of acid-fast origin, but it is added evidence, and is reliable in 90 per cent of cases[6, 7].

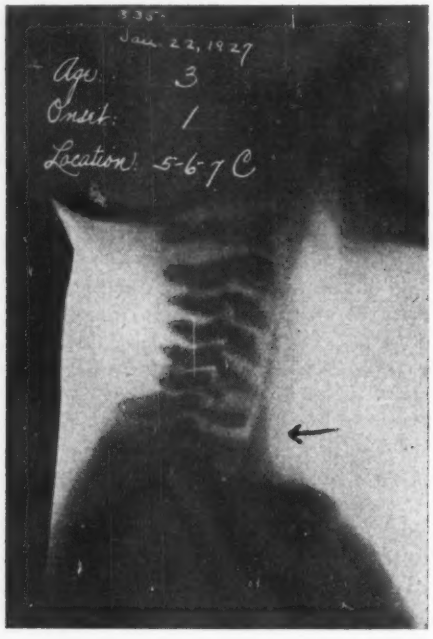
DIFFERENTIAL DIAGNOSIS: A non-tuberculous osteomyelitis, osteitis fibrosa cystica, compression fractures, malignant disease, and syphilis might conceivably be confused with Pott's disease, but all of these are rare in children, and each can be ruled out by careful history, physical examination, the x-rays and routine laboratory procedures.

THE ABSCESS IN POTT'S DISEASE: The abscess developed from the tuberculous spine is of great importance; it helps from the diagnostic point of view, and it is frequently a troublesome complication of the disease. Of the total of eighty-nine cases, seventy had abscesses which were demonstrable by roentgenogram. In twenty-four of the eighty-nine cases there was clinical evidence of the abscess formation manifested by swelling, pressure symptoms, or by draining sinus. Twenty-two of the cases gave both clinical, and x-ray evidence of abscess.

The location of the abscess determined to a large extent the nature of its behavior. When it arose from lesions of the cervical spine, it rarely remained confined to this region, but usually dissected its way into the posterior and superior mediastinal cavities. Symptoms of dysphagia and dyspnea were not uncommonly present from such an abscess due to pressure. In our four cases of cervical Pott's disease, two presented these findings during the acute stage of the disease. Rarely does the abscess from the cervical spine point superficially; the writer has seen one such case in an adult, but never in a child. It is not often necessary to drain the cervical abscesses because under careful conservative treatment they usually become smaller and calcified, and may even disappear entirely. If the abscess points superficially, repeated aspiration is preferred to incision.

The abscess developed from the dorsal spine lesion, as a rule, remains confined to this region, and in none of our cases did it progress to below the diaphragm. In our series of forty-seven dorsal, and four cervicodorsal lesions, no abscess dissected its way through the diaphragm to the lumbar region. The abscess of the dorsal spine, particularly in the upper portion may cause symptoms of pressure and it is felt by some authors[5] that such an abscess does encourage increasing deformity. The abscess of the dorsal spine is usually fusiform in type, but occasionally circular. As a rule, it is visible on either side of the vertebral column in the anteroposterior roentgenogram; it is unlikely to be demonstrable in the lateral view. It dissects its way up and down the spine under the anterior common ligament, usually beyond the limits of the involved vertebrae. Of the forty-seven dorsal, and four cervicodorsal lesions, thirty-three showed abscesses by x-ray, but only four presented clinical evidence of this complication. All of the latter were in the cervical and upper dorsal regions, and gave symptoms of pressure. There was x-ray evidence of abscess in all of the eighteen dorsolum-

bar lesions, and clinically seven were present. In nine of the eighteen which involved the dorsolumbar region, the abscess did go through the diaphragm. Of these, four presented destructive processes extending to and including the first lumbar; three to the second lumbar, and two to the third lumbar. The abscesses of the lumbar spine present a different problem; they are much more apt to become superficial, break down, and form draining sinuses. Such a sinus is always more or less secondarily infected and offers one of the serious complications of the tuberculous lesion. Abscesses from the lesion of the dorsolumbar or lumbar spine commonly follow the course of the psoas muscle, and point to one of three places; the groin, the flank, or the thigh. In thirteen lumbar lesions there was x-ray evidence of twelve, and clinical evidence of ten. There was x-ray evidence of abscess in all three lumbosacral lesions, and clinical evidence in one.



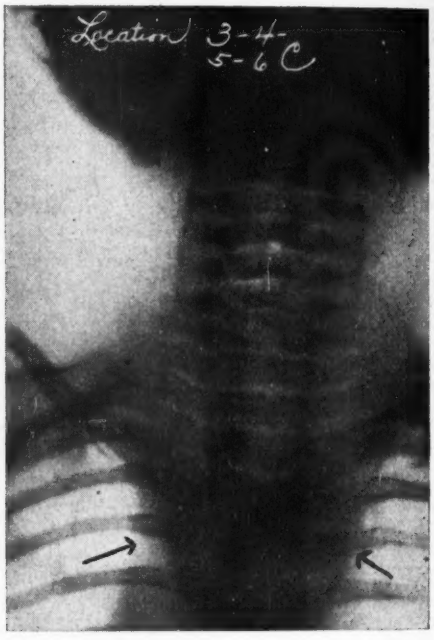


FIG. 4. The Abscess in the Cervical Region.

(a) Lateral view—partially calcified.

(b) Another patient—A-P view, large abscess in superior mediastinum causing pressure symptoms.

TREATMENT: If the case is acute at admission, and because of this does not permit hyperextension, the patient is placed on a Bradford frame; as symptoms become more quiescent hyperextension is gradually started by bending the frame 20° or 30° at the desired level. With further quiescence hyperextension is obtained by the use of plaster shells. The anterior shell is made with the patient supine on Goldthwait irons and in extreme hyperextension. The shell is made to run from the chin to below the knees with the thighs in about 20° abduction to allow for use of a bedpan. When the anterior shell is dry, the posterior shell is made on the patient lying in the anterior shell. With some patients where the lesion is low dorsal, or lumbar, a split plaster jacket (devised by Ghormley) is used. For lesions of the cervical spine, head traction by means of a Sayre sling with the patient on a Bradford frame has been preferred to plaster immobilization. In very few patients have we been able to diminish the kyphos unless we saw the case very early. We think some have been diminished but it is impossible to accurately measure any such change. What we do hope to prevent is an increase in deformity, and to do this we rely chiefly on our hyperextension shells. The child is continuously in one shell or the other and is never removed even for bathing or use of a bedpan. The length of time for the shell treatment depends upon the child's progress clinically and by x-ray. We now feel sure that this period of recumbency and conservative treatment has in some cases been too prolonged. The average period of recumbency for seventy-four cases was thirty-three months. In fourteen cases which came to operation, the average period of recumbency was thirty-nine months.

OPERATIVE TREATMENT: It has been our procedure not to operate upon any child under five years of age, but we are by no means convinced that spinal fusion should not be done before this time. It has so happened that few of our younger patients have been clinically well enough to justify surgery. The factors upon

which we base our decision to do the operation rather than continue with conservative treatment are as follows: First, we must decide whether or not the child will remain well, and not increase its deformity if operation is not done. If it is then decided that fusion is indicated, the following should be fulfilled. (1) Good state of nutrition with a progressive gain in weight. (2) Absence of fever. (3) Normal blood. (4) Absence of pain on passive motions. (5) Absence of muscle spasm. (6) X-ray evidence of healing, or at least, arrest of the progress of the disease. An increase in deformity in spite of hyperextension should indicate operation.

Before operation the patients are allowed up approximately two to four hours daily, wearing a plaster jacket, for a period of six weeks. This is done because patients so treated stand operation better. All of our fusions have been done under ether anesthesia. In eight of thirteen cases the ether has been preceded by avertin.

Our routine procedure is as follows: A modified Hibbs' fusion plus an osteoperiosteal graft has been used in all, and has given uniformly satisfactory results. The incision is made over the spinous processes from the level of two or three vertebrae above the lesion and carried down over the involved region to two or three vertebrae below. The spinous processes are exposed subperiosteally along with the laminae out to the articular facets. Rarely do we destroy the facets, since to do so we feel unnecessarily prolongs the operation. All ligamentous tissue and cartilage over and between the spinous processes are removed. The spinous processes and laminae are "feathered" into numerous pieces and over this bony bed is placed an osteoperiosteal graft which has been removed from the tibia. It is not necessary to suture the graft in place. The periosteum and muscles are sutured as one layer, and the wound is further closed. The patient is allowed to lie in bed on an ordinary mattress for ten days and when soreness has subsided he is returned to his plaster shell.

Each postoperative case has been x-rayed at monthly intervals for six months and we feel that there has been evidence of bone formation at the site of fusion at the end of the second month. This bony deposit increases, and at the end of a year becomes solid. It is probable that at the end of six months these patients could be allowed up with safety, but we have been more conservative and have kept all of them recumbent for one year, and have allowed them up with a long back brace. We still feel that there is danger of the graft bending or breaking unless some external support is worn until puberty is reached. In two of the cases there has been evidence of a break in the continuity of the graft. These areas represent fibrocartilage and should be removed and replaced by bone.

TREATMENT OF THE ABSCESS: The abscess of the cervical and dorsal regions rarely requires intervention even though it may give rather marked symptoms of pressure. It has been our experience that they all quiet down with careful conservative treatment. Calvé[8] has devised a special type of trocar with which he has aspirated abscesses of the dorsal spine, but we have had no experience with this instrument. We are not yet convinced that it is necessary to evacuate these abscesses in order to prevent deformity or insure healing. When the abscesses originating from the dorsal, dorsolumbar, or lumbar regions become superficial, aspiration, but never is incision justified. Aspiration is justified because of two reasons: (1) To attempt to prevent the breaking down of the abscess, and the formation of a discharging sinus, and (2) to gain material for culture and guinea-pig inoculation. Aspiration of the abscess should be done under strict aseptic precautions; it should be carried out in an operating room with most careful technique. A needle sufficiently large should be introduced through good skin and passed obliquely through normal tissue into the abscess cavity. The point of entrance should be away from the dependent portion of the abscess. The abscess cavity is emptied as completely as possible, and repetition of this procedure is justifiable if, and when it refills. Of the eighty-nine cases, fifteen abscesses were aspirated. Ten of these fifteen drained in spite of aspiration. Four drained spontaneously before aspiration. Only one did not drain after aspiration. These figures would indicate that little therapeutic value is attached to aspiration of the abscess but it is still felt that from a diagnostic point of view, it is decidedly justifiable, and if carefully carried out, is a harmless procedure. We have yet to see the value of incision and evacuation of the abscess followed by closure of the wound.

ASSOCIATED LESIONS: Pott's disease is not infrequently associated with tuberculous lesions elsewhere. This is so often true that we routinely x-ray the spine of each tuberculous patient whether or not there is clinical evidence of involvement of the spine. Three "silent" lesions of the spine have been accidentally discovered by roentgenogram supposedly taken to show the hip joints.

In the eighty-nine cases, there were twenty-seven associated nonpulmonary lesions.

END-RESULTS: The eighty-nine cases have been classified as follows:

Excellent, eighteen. In this class have been placed those cases who have maintained a state of good general nutrition and whose local lesion

is healed. Only three of the eighteen had any spinal deformity, and in these instances it was very slight indeed. Five are still under observation in the institution, but are in excellent condition. Two of this group were treated by spinal fusion; the others with jackets and braces only.

Twenty-six cases were rated as *good*. These patients had been well generally and had gained weight but had developed slight spinal deformities as the lesions had healed. Seven of this group are still in the institution. Five of these twenty-six cases had their spines fused at the time of the end-result study, but seven additional cases have been fused subsequently. These last operations have been done too recently to permit end-result studies.

TABLE 3

ASSOCIATED LESIONS IN EIGHTY-NINE CASES OF POTT'S DISEASE

Tuberculosis of Knee	5	
Hip	4	
Ankle	3	
Shoulder	2	
Sacroiliac	2	
Wrist	1	
Elbow	1	
Kidney	1	
Cervical Adenites	5	
Phlyctenular Conjunctivitis	3	
	27	30%

Ten cases were rated as *fair*. All of these had rather marked deformities of the spine; all but one involved the dorsal spine; the lumbosacral junction and sacroiliac region being involved in this case. It has been learned that subsequent to discharge the patient developed a forward slipping of the fifth lumbar on the first sacral necessitating lumbosacral fusion. Prolonged treatment was carried out in every case. Four cases have been fused, and operation refused in one. Two were discharged with rather marked deformities, and one case with moderate deformity; fusion is contemplated in the remaining ones.

TABLE 4

CAUSES OF DEATH IN TWENTY CASES IN SERIES OF EIGHTY-NINE CASES OF POTT'S DISEASE

Amyloidosis	8	(4 died elsewhere)
Pulmonary Tuberculosis	3	(All died elsewhere)
Peritonitis after appendectomy	2	
Shock following spinal fusion	3	(2 died elsewhere)
Tubercular Meningitis	1	
Miliary Tuberculosis	1	
Pulmonary Embolism	1	
Cause Unknown	1	
	20	22.4%

Ten cases were given *poor* ratings because in spite of consistent conservative treatment they developed a progressive deformity and while each is ambulatory and leading a useful life, their deformities are rather extreme. These patients are marked as "hunchbacks". Two of these were fused; two others are still under treatment and will be operated upon before discharge.

The end-results in five cases are *unknown* because it was impossible to trace them.

Twenty of the eighty-nine cases *died* either in the institution or subsequent to their discharge. The high incidence of amyloid disease as the cause of death is consistent with our experience with all types of bone and joint tuberculosis.

END-RESULTS OF OPERATED CASES: Of fifteen cases of spinal fusion done by the end of 1930, thirteen had been performed by the same surgeons in our clinic. Two cases had been fused elsewhere; one obtained an excellent result, the other developed a second lesion below the site of fusion. For this a second operation has subsequently been done. Of the remaining thirteen cases, the results from fusion have been uniformly good.

All obtained solid fusion clinically, but in one there was evidence by x-ray of a defect in the graft. All wounds healed per primum and there were no secondary sinuses. Each patient is ambulatory without an increase in deformity, and the general state of nutrition is good.

Three of the fifteen cases are going without support while eleven are still wearing a brace simply as a precautionary measure. One patient died of postoperative shock.

CONCLUSIONS

1. Pott's disease is the most common form of bone and joint tuberculosis, comprising about 50 per cent of all such cases.
2. Patients with tuberculous joints require specialized institutional care and cannot be satisfactorily treated through an outpatient department, or by ambulatory methods.
3. There may be definite types of the disease demonstrable by roentgenogram and dependent upon the circulation of the vertebral body.
4. The clinical diagnosis is not difficult but the disease can be proved only by biopsy, culture, or guinea-pig inoculation.
5. The disease is most common in the dorsal spine, and it is in this region that the most marked deformities occur.
6. The abscess is important as an aid in diagnosis, and often is a seriously complicating factor.

7. Lesions of the cervical spine usually heal without gross deformity and with a good range of motion under proper conservative treatment.
8. Lesions of the dorsal and dorsolumbar junction frequently develop marked deformity in spite of careful conservative treatment, and it is felt that spinal fusion is the surest means of arresting increasing deformity and establishing a permanent cure.
9. Lesions of the lumbar and lumbosacral regions usually heal with relatively little deformity.
10. In the eighty-nine cases there was a mortality of 22.4 per cent, the most common cause of death being amyloidosis which accounted for eight of the twenty deaths.
11. Fusion of the spine has been carried out in fifteen cases. We have made it a practice not to operate upon any child under five years of age. But we believe that, if the general health of the patient is satisfactory, there is no minimum age at which fusion can be done.
12. The eighty-nine cases were rated as follows:

Excellent	18
Good	26
Fair	10
Poor	10
Dead	20
Unknown	5

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CHEMICAL AND ELECTROGALVANIC BURNS OF TONGUE*

BY WILLIAM J. MACDONALD, M.D.†

Ladies and Gentlemen:

WE have all been faced at times with certain very baffling lesions of the oral cavity. It is quite unnecessary to probe into such pathological entities as pemphigus, lichen planus, syphilis or drug eruptions. They can be extremely bizarre and most certainly are occasionally overlooked.

The greatly increased interest manifested recently by physicians, dentists and physicists has prompted me to select for this address a very specific reason for certain types of oral irritation. Undoubtedly, recognition of its importance has been shown by the awakened interest of physicians and dentists throughout the country. It presents very fascinating possibilities.

We have all been confronted at times with leucoplakia of the tongue in non-smokers, whose blood Wassermanns have been negative. We have seen inflammatory lesions of the gums and buccal mucous membrane, and have failed to arrive at a correct etiological interpretation. I think, therefore, we owe a debt of gratitude to a very astute and exceedingly observant dermatologist of Oklahoma City, Dr. Everett Lain, for pointing out to us the why and the wherefore of certain hitherto unknown oral lesions. If an irritating or potentially irritating substance is applied to the skin or mucous membrane, a dermatitis or stomatitis of varying intensity occurs. Sometimes the offending object is elusive. Outwardly and apparently innocuous, it nevertheless can only have its viciousness dethroned by physical and chemical investigation.

*Read before the American Association for the Advancement of Science, at Boston Medical Library, December 29, 1933.

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CAUSATION

The causes of the type of irritation referred to are as follows:

(1) Imperfect vulcanization of pararubber plates together with the presence of free sulphur and mercury sulphide.

(2) The dissociation of the elemental electrons in metallic dentures in a cavity which by virtue of a potent electrolyte, the saliva, must be regarded as a small galvanic battery. The flow of current is from metals of a higher to those of a lower electropotentiality.

PREPARATION AND ELABORATION OF CRUDE RUBBER

In order to visualize why a toothplate made from rubber can cause trouble, I wish to briefly describe the chemical steps followed in rubber refinement. It will be seen that there are many possibly harmful agents in its elaboration, apart altogether from sulphur used for hardening and mercury sulphide for coloring purposes. Obermayer[1] studied this question very exhaustively. I am indebted to him for my knowledge. The milk juice of the rubber plant is called latex, being composed entirely of carbon and hydrogen. Prior to its use in the rubber industry, latex must be treated in various ways. It is first washed and treated with solvents, carbon bi-

sulphide, benzene, mineral naphtha and carbon tetrachloride. Following this it is subjected to the process of vulcanization, namely heating with sulphur and zinc oxide. Certain chemical substances called accelerators hasten the process of vulcanization. Moreover, to nullify or lessen the property of rubber to absorb oxygen, inhibitors are added. It is clearly seen therefore that various agents can be individually or collectively held responsible for rubber dermatitis. It has been found that the accelerators are predominatingly the offending substances. The best known members of this group follow:

(1) Hexa-nitro-di-phenylamine. This is a powder readily air borne and the cause of numerous cases of dermatitis.

(2) Hexa-methylene-tetra-amine. This substance is intensely irritating upon the skin of workers handling the warm raw rubber product. There is decomposition upon the skin with liberation of formic acid, and this is regarded as the essential irritative factor.

(3) Tri-ethyl-tri-methylene-tri-amine.

(4) Para-phenylene-di-amine. One would expect trouble with this accelerator. Every dermatologist knows its viciousness. Only recently numerous cases of dermatoconjunctivitis from the use of "lash lure" have been reported. "Lash lure" is a toilet article used by women in order to darken the eyelids and eyelashes.

(5) Sapex, a patented accelerator.

(6) B. B. accelerator, a condensation product of butyraldehyde and di-methyl-para-phenylene-di-amine.

(7) Methylenamiline, which often contains traces of uncombined aniline or formaldehyde.

(8) Oxynone.

(9) Phenyl-beta-naphthylamine.

Each member of this group is capable of causing serious skin eruptions, which may be very resistant to treatment.

I have previously enumerated the solvents, of which benzene is the chief cause of trouble.

Zinc, chromium and their compounds, arsenic, guanidine compounds, acetone, ethyl-acetate and sulphur and its derivatives are further chemical substances used in the rubber industry.

Hypersensitiveness to rubber generally is caused by one or several chemicals used in the process of vulcanization. Whenever lesions of the skin or mucous membrane are encountered, a very painstaking differential diagnosis is made. The cases, cited by Lain, Hollander, and those I shall show to-day by lantern illumination have been definitely proved to be chemical or electrogalvanic burns of the mouth. Other possible causative agents have been ruled out.

NATURE OF THE CHEMICAL LESION

Individuals suffering from the effects of undercured dental plates complain of sore mouth, bad breath, a sense of fullness of the tongue, increased salivation, and general discomfort. The tongue itself is perpetually coated and frequently fissured.

It is the consensus that undercuring of the pararubber with sulphur and mercury is the cause of the patient's symptoms. All vulcanite plates are heavily charged with sulphur, which in itself can be and often is an active primary irritant. Vermilion or mercury sulphide, if at all free in the plate, is a second cause of irritation. If the plates are correctly made and properly cured, neither sulphur nor mercury can cause much trouble.

A great deal of work has been and is being done to remove the irritants in vulcanite plates. Other types of plates have been devised. Celluloid, condensite, bakelite, helcolite, alcolite, gold and other metals are being used in the manufacture of newer plates. Unfortunately, they are costly, so that at the present time vulcanite plates are still popular.

ELECTROGALVANIC LESIONS IN THE MOUTH

An electrogalvanic burn in the mouth is an acute or chronic type of stomatitis or an allied pathological state, caused by dissimilar metal dentures constituting a galvanic battery, the saliva acting as an electrolyte. This may sound fantastic. However, it is really necessary to realize that it is not by any means a figment of the imagination. At the A. M. A. convention in Philadelphia in 1931, and New Orleans in 1932, it was my privilege to watch Dr. Everett Lain demonstrate the solidity of my statement. He used a Weston direct current micro-ammeter number 320. To this, suitable electrodes with copper contact points were connected. That electrode coming from the positive side of the micro-ammeter is placed upon the negative metal, for instance gold. The other electrode makes contact with the positive metal, possibly one containing zinc or aluminum. With the mouth open the meter needle ascends to its highest reading and gradually returns to zero when the mouth becomes dry. That is to say, the current ceases to flow when the electrolyte, in this case saliva, is withdrawn or dries up. It is to be noted that the intensity of the current depends not only upon the metallic nature of the dentures, but also upon the chemical reaction one finds in the mouth. Salivas which are hyperacid or hyperalkaline are more liable to cause irritation. A greater surface of metallic work naturally will cause grosser lesions. J. D. Hodgen in Practical Dental Metallurgy, seventh edition, gives the metals in electromotive force series.

METALS IN ELECTROMOTIVE FORCE SERIES

Positive End	
Rubidium	Cobalt
Potassium	Nickel
Sodium	Tin

Lithium	Lead
Barium	Hydrogen
Strontium	Copper
Calcium	Arsenic
Magnesium	Bismuth
Aluminum	Antimony
Manganese	Mercury
Zinc	Silver
Chromium	Palladium
Cadmium	Platinum
Iron	Gold

Negative End

From what has been said, it can be realized that dissimilar dental fixtures, be they plates, amalgams, crowns, or bridges, cause trouble of a more or less severe nature in accordance with the distance that separates them in the series. Restorative amalgams consist of three or more of the following metals: silver, tin, zinc, copper and sometimes gold or platinum, to which mercury is added to cause successful amalgamation. Gold used in dentures is alloyed with one or more of these metals, copper, nickel, silver or platinum. Copper is used for hardening, and the others for color and strength.

SYMPTOMS

I. Objective Signs in the dentures.
II. Objective Signs on the mucous membrane.
III. Subjective phenomena.

1. The constant dissociation of the electrons in the metallic dentures brings about a migration of electrons from the higher electromotor series of metals to the negative end. This simply means that such metals as gold and silver become discolored, at times eroded, and certainly loosened. Gold fillings have an uncanny way of falling out. Possibly upon poor technique can be laid some of the blame. I prefer, nevertheless, to believe that the real cause is this constant electrogalvanism in the mouth. Why is it that teeth become discolored? Tobacco and poor hygiene cannot be entirely blamed. I have seen inveterate smokers with white teeth, a state of perfection I ascribe to their freedom from dissimilar metal replacements. I firmly believe that some of the blame for discolored teeth, and much of the blame for discolored fillings should be laid to that metallic potpourri we find in the mouth of the twentieth century human being.

2. Individuals suffering in this way present quite a medley of irritations. The tongue can not only be fissured most painfully and to a considerable depth, but it is swollen and continually coated. As time goes on leukoplakia replaces the initial irritation. Rarely but occasionally are seen black tongues. I hope to show you one later on. The lingual papillae are hypertrophied. The borders of the tongue present indentations corresponding to the offending dentures.

Upon the buccal mucous membrane, lesions of a more or less similar nature are noticed. Thin white or grayish irregularly shaped discoloration, occasionally dark pigmentation, leucoplakia or ulceration are noticed. The earliest lesions would naturally be purely inflammatory.

3. The subjective signs are many and varied. Possibly the commonest is bad breath. I wonder how many people possessing bad breath have questioned the efficiency of their entire gastro-intestinal tracts when the blame could be found at the very doorway. A metallic taste is quite common, together with a sense of fullness in the mouth. The flow of saliva increases, or at times there is a marked feeling of dryness in the throat. Individuals who smoke complain of burning tongue. Those who never smoke also state that their tongue is easily irritated by simple foods. The tongue appears to be hypersensitive. An occasional electric shock is complained of if the dissimilar metals make contact.

As times goes on these patients acquire a tolerance for their discomfort never realizing its causation. It is treated quite uselessly with stomachics, diet or a mouthwash. If the irritation is severe enough and the sufferer constructed according to a nervous pattern, there follow indigestion, loss of weight, anemia and general ill health.

SUGGESTIVE REMEDIAL MEASURES

Patients whose symptoms can be, undoubtedly, traced to either chemical or electrogalvanic burns should be urged to unify their fillings. The great and obvious objection of course is expense. It is not always feasible to indulge in gold plates or unified gold inlays or fillings. Drastic measures would be called for in severe cases where leukoplakial degeneration might lead to carcinoma.

Dentists should, when possible, use gold or silver entirely. Moreover the patient should be told of the possibility of oral trouble later on should he demur at treatment on the score of expense.

It is satisfactory to note that when well vulcanized plates are substituted for those at fault the patient usually recovers. When, too, the offending dentures are removed, and others of uniform electropotentiality substituted, there is positive and rapid relief.

In conclusion I wish to briefly report five cases of electrogalvanic or chemical burns of the tongue:

CASE 1. H. J. C., aged fifty-seven; smoker; seen for first time on May 7, 1933. Patient was referred for treatment of eczema. During the course of the usual physical examination, the tongue was seen to be markedly leucoplakial. Sore throat and pharyngeal dryness accompanied the objective and major phenomenon. Increasing debility, vertigo and constipation were the only subjective signs that could be elicited as a cause of the eczema. The latter responded satisfactorily to roentgen ray therapy. The

Wassermann was negative. This man's lower back molar teeth were filled with amalgam. Several years later he had a gold upper plate made to replace several extracted teeth. Since this restorative plate was worn his throat became dry and the tongue

CASE 1. Leukoplakia lingualis. Electrogalvanic burn.

leucoplakial. It was impossible to get accuracy as to dates in this case, but emphasis was laid on his own accurate observation.

On April 3, 1934, I saw this man again. At this time his leukoplakia had improved sixty per cent and his subjective oral symptoms had vanished. The gold plate had been replaced by a non-metallic vulcanite plate.

Diagnosis: Electrogalvanic burn of tongue with marked leukoplakia.

CASE 2. W. J. McC., aged twenty-nine; male; smoker. This man was referred by Dr. Carl DePrizio suffering from "black tongue". His physical examination was essentially negative. His Wassermann was

CASE 2. Pigmentation and electrogalvanic burn of tongue.

negative. In October, 1932, this man suffered from Vincent's disease associated with severe angina and loss of several teeth. In January, 1933, the gums and mouth had recovered to such an extent that restorative work was undertaken to replace the lost teeth. This work was executed with gold. The patient, in addition, has eight silver or amalgam fillings. Prior to the restorative gold work the tongue was normal, and subjectively the patient felt quite well.

About February, 1933, i.e., one month later, he noted:

(1) Fullness of the tongue and increasing pigmentation, black in color.
(2) Subjective signs such as fatigue, sleepiness, anorexia.

On examination there was observed:

(1) Three gold inlays, i.e.,
  (a) Left upper outer incisor joined by a fixture to the first bicuspid.
  (b) Second left lateral bicuspid.
  (c) Second right lower bicuspid.
(2) Elongated lingual papillae, all being markedly discolored black.
(3) The buccal mucous membrane was not discolored black, but was covered with a blue opaline membrane.
(4) A deep median fissure running back to the circumvallate papillae.
(5) Numerous hypertrophied lingual papillae.

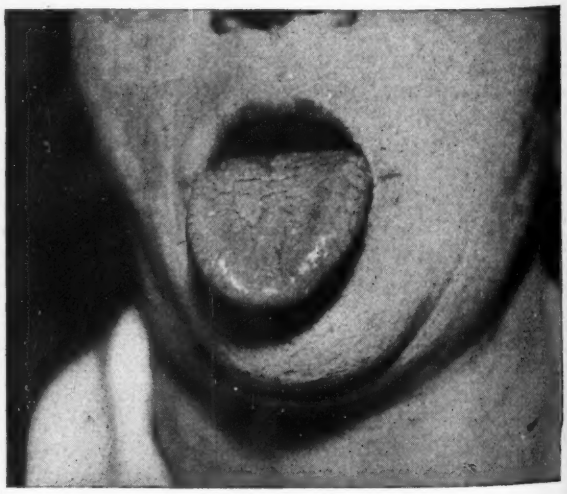
The incidence of the actual subjective phenomena, and the striking objective lesions pointed to electrogalvanic burn of the tongue.

Scrapings of the tongue for mycelia were negative.

Diagnosis: Electrogalvanic burn of tongue.

CASE 3. B. L., aged sixty-three, female: The chief complaint of this patient was generalized eczema. A careful history was taken and all possible causes of the skin disease gradually eliminated. However, one month prior to the onset of the dermatological condition, she had visited a dentist. Here she was fitted with a low-priced, ill-fitting metallic plate, which has never been comfortable. She was advised to discard the plate and be refitted. It might be argued that the plate had nothing to do with the incidence of a marked skin disease. I can only say that as one followed the other so closely, it is reasonable to assume this is a case of eczema caused by metallic absorption.

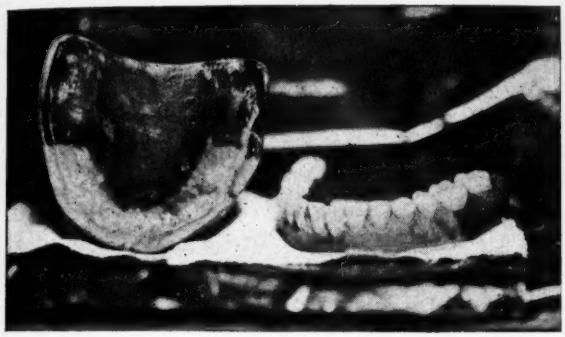
CASE 4. J. Z., aged fifty-one; female: This patient presented herself in May, 1933 for treatment of "sore mouth and tongue". She was an active woman,



CASE 4. Chemical burn of tongue and mouth from a badly made vulcanite plate.

and careful physical examination was essentially negative. Blood Wassermann was negative. Tongue scrapings were negative for mycelia. She very emphatically stated that her tongue, mouth and gums were uncomfortable and inclined to burn. Her tongue was markedly fissured. Early leukoplakia showed very definitely.

These symptoms dated back nine years at which time she was fitted with a heavy, gray-colored tooth plate. This plate has never been comfortable. It appeared to be made of black vulcanite, very likely improperly cured.



CASE 4. Tooth plate made of black pararubber, sulphur and vermilion, and causing fissured tongue, early leukoplakia and marked discomfort and burn.

I suggested a new and better plate.

In March, 1934, I saw this patient again. My advice had been followed. Her subjective symptoms had vanished. The fissures on her tongue had smoothed out and there was no leukoplakia. She felt completely relieved.

Diagnosis: Chemical burn of tongue.

CASE 5. J. K., aged thirty; female: This patient was referred to me with a small dime-sized, white, moderately infiltrated lesion on her tongue opposite the upper left first bicuspid. This lesion appeared five months previously at which time the left first bicuspid was filled with silver. I referred her to her dentist. He removed the filling and replaced it with gold. She had other gold fillings in her mouth, but no silver fillings. I saw this patient early in 1933, at which time the lesion had disappeared, and the fillings in her mouth were all of the same electropotentiality.

Diagnosis: Electrogalvanic burn of tongue.

CASE 5. Electrogalvanic burn of tongue from a dental filling.

CONCLUSION

(1) Attention has been drawn to a definite and clear-cut type of stomatitis and glossitis. It is believed "under or over-cured" tooth plates or dissimilar metallic dentures can be the cause of such symptoms.

(2) Before a diagnosis is made it is essential to eliminate such possible causes as syphilis, lichen planus, carcinoma, over-indulgence in tobacco, food allergy, trauma, anemia, Vincent's disease, drug eruptions and certain other definite oral diseases.

(3) The removal of the cause is usually followed by improvement or recovery.

(4) The speaker wishes most emphatically to draw attention to the early work of Dr. Everett Lain on this subject. He also has quoted very freely from Dr. Lain's publication.

REFERENCE

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A COMPARISON OF SKIODAN AND DIODRAST AS VASOGRAPHIC MEDIA, WITH SPECIAL REFERENCE TO THEIR EFFECT ON BLOOD PRESSURE*

BY EDWARD ALLEN EDWARDS, M.D.,† AND FERNANDO BIGURIA, M.D.†

IN a recent communication[1], was discussed the present status of vasography, the visualization of blood vessels by the injection into them of opaque media and the making of a roentgenogram. It was pointed out that we have now a safe method of making good roentgenograms of both arteries and veins. The following criteria were laid down for the judging of a vasographic medium.

1. It must be satisfactorily opaque, even when diluted to some extent by the blood.
2. It must be freely miscible with the blood and not give embolism, or false filling defects in the roentgenogram.
3. It must be sufficiently limpid to be introduced into the vessel quickly, and through a needle small enough to enter without undue damage.

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4. It must not injure the blood chemically, i.e., not give coagulation or hemolysis.
5. It must not irritate the blood vessels sufficiently to give thrombosis through intimal damage, or endanger an already anemic limb through additional vasospasm.
6. Its accidental escape outside the vessel should not cause severe inflammation.
7. It must not, in the doses used, have a toxic effect on the general organism, exclusive of the blood or blood vessels.
8. Its use must not be so painful as to necessitate a general anesthetic.
9. It must not be appreciably altered by adequate sterilization.

At the time of that communication, Skiodan, in 40 per cent aqueous solution, was considered as best fulfilling all these criteria. It is described as a brand of methiodol, "mono-iodomethane of iodine, in firm combination". The 40 per cent solution is sterile and stable, casts a good shadow, is freely miscible with blood and is easily injected. It is excreted by the kidneys, rendering the urine opaque to the x-rays, and allowing good roentgenograms to be made of the urinary tract. It is almost entirely excreted in two hours. The adult dose for intravenous urography is 50 cc.

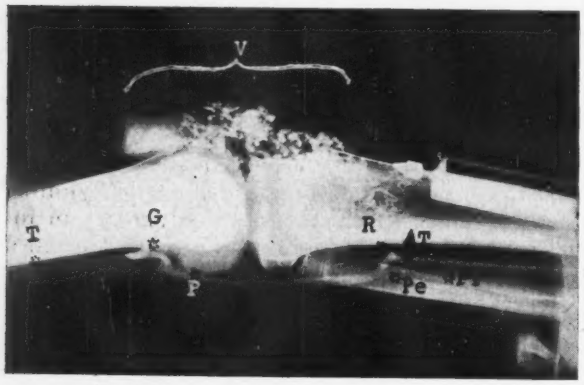

FIGURE 1. Arteriogram of a Congenital Arteriovenous Fistula. Lateral view of the affected limb.
A tourniquet 'T' was applied around the lower third of the thigh, tight enough to shut off the arterial flow. Thirty cc. of 35 per cent Diodrast were then injected into the anterior part of the vascular tumor. Note the large undifferentiated plexus of vessels 'V' fed by one vessel 'G', corresponding to the lateral superior genicular artery; and a second vessel 'R' corresponding to the recurrent tibial branch of the anterior tibial artery. 'P' is the popliteal, 'AT' the anterior tibial, 'PT' the posterior tibial, and 'Pe' the peroneal artery.

Later, Diodrast in 35 per cent solution was tried as a vasographic medium. It was introduced by the Winthrop Chemical Company as a urographic medium with an adult dose of 20 cc. They describe it as "3.5 diiodo—4—pyridon—N—acetic acid diethanolamine, containing about 50 per cent of iodine in firm combination. The aqueous solution is neutral, clear, and nearly colorless. It is very stable and can be sterilized by heat, in the customary manner, without decomposition".

Considered as a vasographic agent, Diodrast seems at least as good as Skiodan. In addition it has certain advantages to be mentioned later. In 35 per cent solution it casts a shadow as opaque as the 40 per cent solution of Skiodan. (Figure 1.) As noted, it is also freely miscible with blood; it is limpid; it does not damage the blood or blood vessels and it does not cause severe inflammation if it escapes outside the vessel wall. There are, however, two points of divergence between the two substances; first, in the production of pain; secondly, in their effect on blood pressure.

Skiodan, on quick injection, gives some cramping pain. While this is hardly noticeable on intravenous injection, it is at least moderate on intra-arterial injection. In this latter case, too, it produces an involuntary contraction of the muscles of the part, both in the dog and in the human being. Diodrast, on the contrary, produces neither the slightest pain nor muscular contraction, whether it be used in the artery or the vein.

The first intimation of a blood pressure effect of either substance came in the course of an arteriography with Skiodan in a dog. A very prompt rise in blood pressure was obtained, which was so high as to resemble the effect of adrenalin. On trying the injection of Skiodan intravenously, a similar but less marked rise of blood pressure was noted. (Figure 2.) This corresponds to the results reported by Heathcote and Gardner[2].

The effect of an intravenous injection of Diodrast in the dog was then tried. A fairly prompt *fall* in blood pressure was obtained. (Figure 3.) The animal experiments with these two substances were carried out in collaboration with Dr. E. Ginsburg and Dr. M. Kozodoy in the Physiology Department of Tufts College Medical School.

Blood pressure readings were made of patients receiving intravenous injections of these two substances. The response was in every way similar to that in the dog. Figure 4 shows the blood pressure chart of a patient after the intravenous injection of 20 cc. of Skiodan. There is noted, two minutes after injection, a *rise* in blood pressure, reaching a maximum four minutes after the injection. These time elements as well as the amount of rise is rather typical. For Skiodan, a systolic rise up to 20 mm. Hg. and a diastolic rise of 8 to 10 mm. Hg. is the rule.

Figure 5 shows the blood pressure chart of a patient receiving an intravenous injection of 20 cc. of Diodrast. The *fall* in blood pressure can be seen to start one minute after the injection, reaching its maximum at five minutes, and recovering its original level at fifteen minutes.

For Diodrast, a systolic fall of 15 to 20 mm. Hg. and a diastolic fall of 8 to 10 mm. Hg. is the rule.

From the experimental data so far obtained, patients with arteriosclerosis, hypertension, or aneurism would seem to be poor subjects for the use of Skiodan, because of the danger, when the blood pressure rises, of the rupture of a blood vessel or of the aneurism. Conversely, Diodrast would seem to be the ideal medium in these cases. As for patients with heart disease, the choice between these two substances will depend on a knowledge of the exact mechanism involved in the blood pressure changes.

The dose of Diodrast to give good urographic pictures in an adult is 20 cc. Nevertheless, we have twice used 30 cc. in a boy of 16 (figure 1) with no untoward effect. That this larger amount is not always a safe dose, however, was indicated by the results in one patient. Fifteen minutes after the injection of 30 cc. of Diodrast into his femoral artery, the patient complained of pain along the course of this vessel, and the limb became cold. In another twenty minutes this was superseded by a definite reactive hyperemia. Later, this patient had nausea and vomiting and showed a slightest possible trace of albumen in the urine. This is the first time we

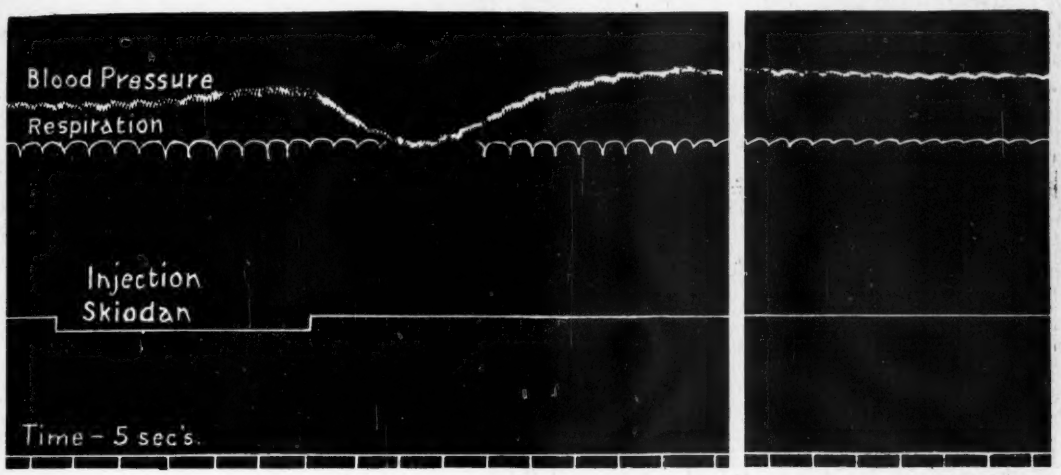

FIGURE 2. The Blood Pressure Effect of Skiodan in the Dog.

For lack of space that part of the record is left out intervening between the beginning of the effect on the left, and ending with its disappearance on the right.

The dog was a male weighing 14.5 Kg. Twenty cc. of a 40 per cent solution were injected intravenously. The blood pressure showed a prompt primary rise from 96 to 102 mm. Hg., then 15 seconds later, a fall to 80 mm. Hg., and then a slower secondary rise to 112 mm. Hg. This was fairly well sustained, gradually returning to normal in 9 minutes and 10 seconds after the end of the injection.

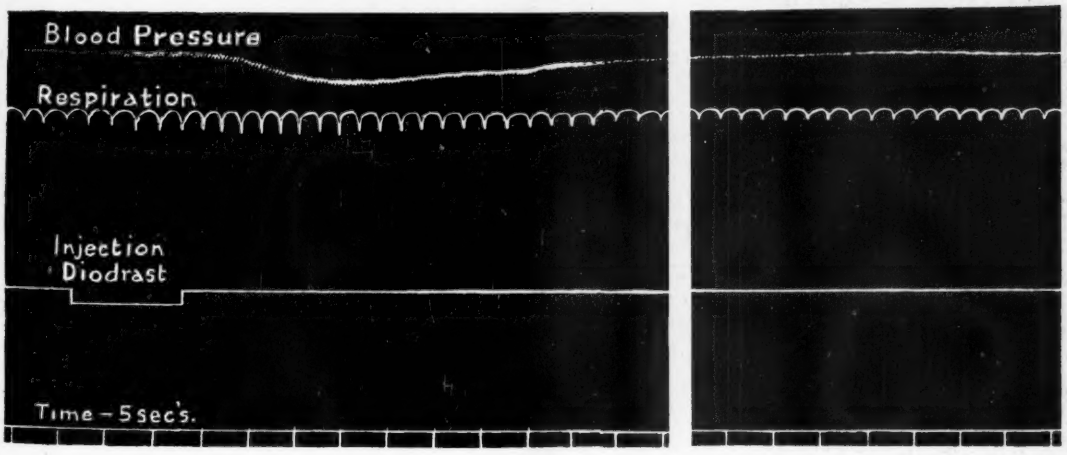

FIGURE 3. The Blood Pressure Effect of Diodrast in the Dog

Same dog as in figure 2.

Ten cc. of a 35 per cent solution of Diodrast were injected intravenously. The blood pressure showed a fall 20 seconds after injection from 106 to 97 mm. Hg. and was normal in 6 minutes and 30 seconds.

have seen either of these results, i.e., arterial spasm or albuminuria after the use of Diodrast.

There are some cases in which it is more obvious that the amount of Diodrast necessary to fill the vessels is more than can be constitutionally borne. The following case illustrates such a dilemma: a child of 2½ years was referred for visualization of a large congenital

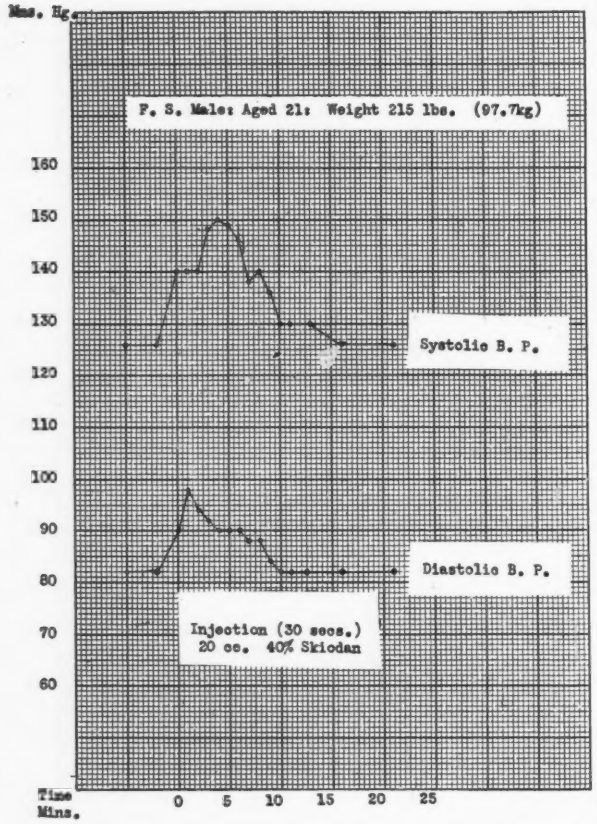

FIGURE 4. The Blood Pressure Effect of Skiodan in the Human Subject.

angioma of the neck.* The angioma extended from the right angle of the jaw to the sternal notch. In its lower extent it filled, at each deep expiration, with about 25 cc. of blood, and emptied itself at each deep inspiration. Altogether it contained, when engorged during deep expiration or crying, an estimated 50 cc. of blood. The visualization was done under primary ether anesthesia. Ten cc. of Diodrast were injected into the angioma. The vasogram obtained was poor, first because of the dilution of the medium by the large amount of blood present in the venous sac; and secondly, because of the very active tidal "wash" in it. Furthermore, the child seemed quite pale after the injection and the pulse was rather small. It was thought dangerous therefore to use a larger dose of Diodrast. The procedure was repeated, using 20 cc. of Skiodan injected into the angioma. A fairly good roentgenogram was in this way obtained. The child showed no constitutional reaction whatever from the use of the Skiodan.

*This patient was referred for vasography by Dr. A. R. Kimpton of Boston, who later successfully removed the angioma.

CONCLUSIONS

1. Diodrast casts a good shadow on the roentgenogram, while producing no local or general bodily effect other than a fall in blood pressure. It is therefore the best available agent for vasography. It is further superior to Skiodan in that its injection is painless, where-

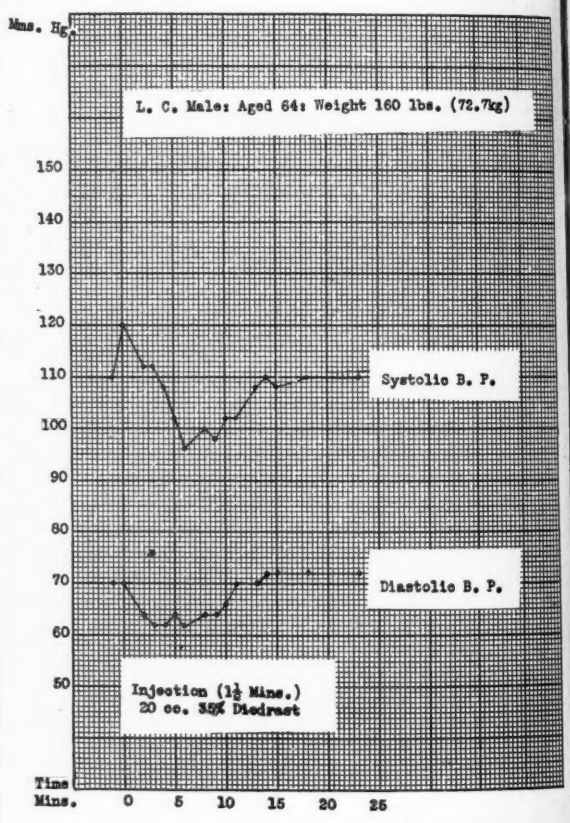

FIGURE 5. The Blood Pressure Effect of Diodrast in the Human Subject.

as the injection of Skiodan produces some pain and muscular contraction.

2. Experimental work in dogs and clinical observation in human beings have shown that after the intravascular injection of Skiodan there is a rise in blood pressure high enough to be important; and that after the injection of Diodrast, there is a considerable *fall* in blood pressure.

3. Because of these blood pressure effects Diodrast would seem to be the agent of choice in patients with hypertension, arteriosclerosis or aneurism; while Skiodan would seem to be contraindicated in such patients. Our opinion on the choice of these media in patients with heart disease will have to wait until we know

the exact mechanism of the blood pressure changes.

4. Skiodan is obviously preferable in severe hypotension. Also, because the maximum dose of Diodrast for infants or children is some fraction of 20 cc. (the adult dose) Skiodan is preferable in these patients when there are present large blood vessels, requiring much of the vasographic agent.

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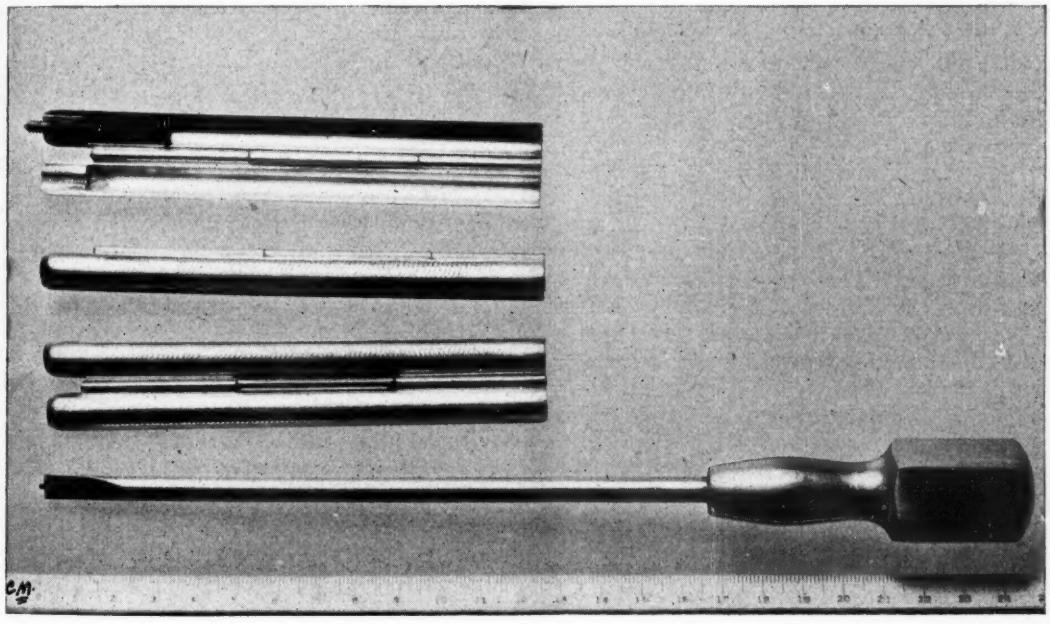
2 Heathcote, T. St. A., and Gardner, R. A.: Abrodil: An experimental investigation. Brit. J. Radiol. 4: 641, 1931.

A BONE PLATE SCREW HOLDER*

BY A. WILLIAM REGGIO, M.D.†

IT has been the experience of the writer when plating a fractured femur, or other bone, where screws an inch or more in length are required, that with the Pilling-Sherman screw driver an annoying "wobble" of the screw is apt to occur, before the screw engages firmly in the drill hole.



This common experience has very probably also annoyed many other men.

Thus, in an effort to overcome this "wobble", the writer devised the simple instrument here shown.

The diameters of the screw driver and screw holder are the same as that of the head of the Standard Metal Sherman Bone Screw. The thread in the "nose" of the holder is the same as on the standard screws. Thus the screw to be inserted is held firmly and no side-play or wobble can occur.

This preliminary "wobble", besides being so annoying, also damages the sharp edge of the drill hole in the bone and is apt to cause the screw to be inserted off-center, as well as widening the hole slightly.

The mechanics of using the instrument are so obvious, that no detailed description is necessary.

With the screw in place, as shown, the holder is closed, the tip of the screw inserted through the hole in the plate, engaging the drill hole beneath it. The screw driver is then inserted and cannot help engaging the slot in the screw.

When the screw is firmly engaged in the bone, the holder (or sheath) is allowed to open and is discarded.

The device is simple and efficient. There are no parts to get out of order, and it is inexpensive.

*From the Fracture Service of the Massachusetts General Hospital.

†Reggio, A. William—Surgeon to Out-Patients, Massachusetts General Hospital. For record and address of author see "This Week's Issue," page 599.

CASE RECORDS
of the
MASSACHUSETTS GENERAL HOSPITAL

ANTE MORTEM AND POST MORTEM RECORDS AS USED IN WEEKLY CLINICAL-PATHOLOGIC EXERCISES

EDITED BY RICHARD C. CABOT, M.D.

CASE 20391

PRESENTATION OF CASE

A sixty-four year old Canadian contractor entered the Baker Memorial complaining of pain in his chest of two days' duration.

He was perfectly well until two days before entry, when following a day spent to some extent outdoors he noticed the sudden onset of pain in the left lower anterior chest. The pain was accompanied by a barking non-productive cough which greatly aggravated it. He felt feverish at times but had no shaking chills. He felt slightly asthmatic and not up to par. He went to bed and stayed there. Twenty-four hours after the onset of these symptoms a physician advised admission to a hospital. He had no cold and no other symptoms preceded this present attack.

His family and marital histories are irrelevant.

He had lived in Massachusetts for the past twenty-five years. He had had the usual childhood diseases and a severe attack of scarlet fever in infancy. There was no history of diphtheria, rheumatic fever or chorea. He had an attack of pneumonia followed by pleurisy at the age of thirty-two. There was a question of tuberculous infection at that time and he remained in a sanitarium for three months, although he was never told definitely that he had tuberculosis.

Physical examination showed a well-developed and well-nourished man, rather flushed, and uncomfortable because of pain in the left lower chest on moderate inspiration. His color was good. There was slight injection of the conjunctivae. The heart was not enlarged. The sounds were only fair. There was a soft systolic murmur. The blood pressure was 80/35. The peripheral vessels were thickened and rather tortuous. The chest showed slight dullness in the lower left axilla and at the left base. The breath sounds over this area, especially in the axilla, were rather harsh, with some prolongation of expiration. Breath sounds and tactile fremitus over the anterior lower left chest were somewhat diminished. There were a few fine and medium râles low in the left axilla and at the left base after coughing. There was no pectoriloquy. The abdomen was slightly distended but soft. There was very slight tenderness just above the costal margin in the right upper quadrant.

The temperature was 101°, the pulse 100, the respiration 30.

Examination of the urine showed a specific gravity of 1.024. There was an occasional white blood cell. Examination of the blood showed a red cell count of 4,900,000, a hemoglobin of 80 per cent, and a white cell count of 40,000, with 94 per cent polymorphonuclears. Examination of the sputum showed a pneumococcus which did not type as I, II, or III.

A portable chest film showed dullness involving the entire area in the region of the left upper lobe. The apex was completely obliterated by the dullness and the lower extremity of the dull area extended to the eighth rib in the right axillary line. The left side of the diaphragm was elevated. The heart was enlarged and slightly displaced to the left.

The day following admission his temperature rose to 104.8°, the pulse to 110, and the respirations to 37. The blood pressure was 102/50. The respirations were irregular and rather shallow. He was drowsy throughout that day but could be roused. He was put into an oxygen tent that night with some relief of cyanosis which had developed. Examination of the chest showed diminished breath sounds at both bases with bronchovesicular breath sounds in the left interscapular region. The number of râles was increasing. The abdomen was slightly more distended but still fairly soft. A film of his chest taken twenty-four hours after the first one showed beginning consolidation in the right upper lobe. A film taken the following day showed increase in the size of the dull area in the right lung, but not much change in the size of the dullness on the left side. The only normal aërated lung appeared to be in the right apex and infraclavicular region. His white cell count came down tremendously, to 12,900, with 92 per cent polymorphonuclears. He rapidly became worse and died four days after admission.

DIFFERENTIAL DIAGNOSIS

DR. WILLIAM H. SMITH: Pain in the left chest in a man of sixty-four, the arteriosclerotic age, would make me think of angina pectoris; the pain associated with cough raises the question of pulmonary infection, a pleurisy secondary to pneumonia. Pain on exertion, or under emotional strain, would suggest angina pectoris. One can have attacks of angina pectoris in the absence of exertion or emotional strain. Nocturnal angina, waking the patient from sleep, is, in my experience, due to a high diastolic pressure. If the pain in this case was associated with inspiration, it would accentuate the possibility of pleuritic infection, possibly pneumonia.

"This pain was accompanied by a non-productive cough which greatly aggravated it." That is not the history of angina pectoris as a rule. It does not definitely state that the onset

of the pain was sudden, as usually is true in angina pectoris. One could not exclude, from the history, an infarct associated with pulmonary edema. The associated temperature of 101° makes acute pulmonary infection most likely. It is possible in elderly people to have frank pneumonia with very little temperature. Every elderly patient with bronchitis, even with slight temperature, should be carefully examined for pneumonia. He had an attack of pneumonia, and there was a question of tuberculosis. It is often difficult in bronchopneumonia at the apex or in slowly resolving areas of pneumonia to exclude tuberculosis. Oftentimes an accurate history of the onset of pneumonia, sudden or gradual, with or without chill or bloody sputum, and the duration of the fever, will help throw the diagnosis into the acute pulmonary infection group. This is especially true in the pneumococcus group. Occasionally actinomycosis of the lung or fungus infections will simulate tuberculosis. If the patient was well nourished at sixty-four the tuberculosis, if present, apparently had not sapped his nutrition. It is very difficult in the presence of a pulmonary condition to interpret accurately a cardiac condition. There are so many râles that the noise made by the patient's breathing tends to obscure murmurs. Even pericarditis may be overlooked under these conditions.

The blood pressure of 80/55 is unusual for a man of sixty-four. With such a low blood pressure and a doubtful history of tuberculosis, one would have to think of the possibility of Addison's disease. I am accustomed to interpret breath sounds in terms of vesicular, bronchovesicular, or bronchial. The lung signs suggest consolidation with possible pleurisy. The presence of slight tenderness just above the costal border on the right side would make me think possibly of gall bladder or high appendix. Spasm is not noted.

The specific gravity of 1.024, with absence of sugar, is high and certainly does not suggest any chronic kidney condition. The white blood cell count of 40,000, with 94 per cent polymorphonuclears, suggests pneumonia or pus.

The chest showed involvement in the region of the left upper lobe. Place your physical signs first; if they do not agree with the x-ray findings, check with your x-ray man. Also check your physical findings with your laboratory controls; if they do not agree, talk it over with the man who is responsible for the laboratory reports.

Am I going too far, Dr. Mallory?

DR. TRACY B. MALLORY: No. I believe in that.

DR. SMITH: The blood pressure was 102/50. I admire a man who can take a blood pressure within two points. That is a degree of accuracy I do not think important. It is a question of hyper-pressure or hypo-pressure and the difference of one or two points is negligible.

It seems to me the whole question in this case lies between tuberculosis with pleuritic effusion, or acute pneumonia with fluid or an atelectatic lung. Of course there is the possibility of infarction with secondary abscess. No etiology exists for infarct. The high white cell count favors pneumonia. Are there any questions?

A PHYSICIAN: Would the blood pressure of 80/35 be due to complicating coronary disease?

DR. SMITH: It might be, but the picture is not that of coronary disease. The essential symptoms of coronary disease with infarct are severe and constant pain of the angina type, slight fever, slight leucocytosis, falling blood pressure, with oftentimes a pericarditis, possibly later an embolus from the associated mural thrombus.

CLINICAL DIAGNOSIS

Lobar pneumonia.

ANATOMIC DIAGNOSES

Lobar pneumonia.
Pericarditis, acute fibrinous.
Hydrothorax, bilateral.
Pleuritis, chronic fibrous.
Chronic vascular nephritis.
Arteriosclerosis: moderate coronary; slight aortic.
Diverticulosis of sigmoid.
Diverticulitis of sigmoid with perforation and abscess formation.

PATHOLOGIC DISCUSSION

DR. MALLORY: We found a lobar pneumonia which apparently started in the left upper lobe and then extended into the right lung and portions of both the right upper and right lower were involved. The involvement there was younger, more recent consolidation than the original left upper lobe process. There also was a massive collapse without consolidation of the right lower lobe. I think usually these cases with a great deal of consolidation are unlikely to get collapse of what little lung is left; a compensatory emphysema is more usual.

There were a few other findings, as one would expect in a man of sixty-four. There is almost always a great deal more pathology in an old than a young man. The most important thing was a marked diverticulosis of the sigmoid. One diverticulum had become inflamed and perforated. There was a small abscess, completely walled off, which had evidently appeared weeks or months before, and there was nothing in the history to suggest it.

The very low blood pressure in the case interested me. The point that one of Dr. Smith's former pupils, Dr. Robert Loeb, is developing at the present time I am sure has a certain amount of truth in it. He believes that a condition of shock, of low blood pressure due to blood stagnation in the portal areas and lack of blood

in the systemic circuit, which we know is very common postoperatively, is also frequently present in infectious disease. The very low blood pressure in certain cases of pneumonia he believes is essentially due to shock rather than to cardiac failure.

CASE 20392

Presentation of Case

The patient was a thirty-seven year old American baker. He had been in an automobile accident eight months before admission and had not been well since. He had had some swelling of his abdomen for the past six months, and had been slightly jaundiced on several occasions. During the past several months he had lost about fifteen or twenty pounds in weight. He had worked in a bakery until four days before admission, when the abdominal swelling and jaundice became much worse. At about midnight on the evening before admission he experienced a sudden severe pain throughout his abdomen. He felt nauseated but could not vomit. The pain persisted to a less degree until admission. He passed several small watery stools during the following day. The abdominal swelling had increased.

There was no history of alcoholism.

Physical examination showed a very ill, pale, restless, and dyspneic man. Definite jaundice could not be determined because of artificial light. He complained of pain in his abdomen and of great thirst. The pupils were equal, regular, and reacted normally. His sclerae were injected and probably icteric. The mouth was dry, the pharynx clear. Breathing was rapid and deep. The lungs were clear. The heart was displaced slightly upward, but apparently not enlarged. The action was regular and rapid, rate 130. The sounds were of normal quality. No murmurs were heard. The blood pressure was 118/80. The abdomen was markedly distended with fluid. There was a round area of red discoloration around the umbilicus. There were no areas of localized tenderness. No masses or abdominal organs were felt. There were no enlarged superficial veins.

The temperature was 100.6°. The respirations were 48.

Examination of the blood showed a red cell count of 4,700,000, with a hemoglobin of 60 per cent. The white cell count was 2,500, with 68 per cent polymorphonuclears. The icteric index was 50.

He rapidly failed, his breathing became very stertorous, the blood pressure fell to 80/40, the pulse became too weak to be obtained at the wrist, and he died on the day following admission.

Differential Diagnosis

Dr. Chester M. Jones: The only facts that we have are that he was a young man of thirty-seven and that he was in an automobile accident eight months before he came in. He had not been well since then. He noted that for six months his belly had been swelling and he had lost weight during that time. He also had noticed slight jaundice on occasions without any association of pain or other symptoms, so far as we can tell from the history. With that in the background we assume that there is a condition in the belly which is causing ascites—at least that is the proper assumption—and that it is associated with jaundice. Whether it has anything to do with the automobile accident I do not know. I cannot imagine anything that would permit him to work for eight months and yet produce ascites and jaundice, so I think that ought to be disregarded.

With ascites and jaundice for eight months one has to think of intrahepatic disease of some sort, and certainly an intra-abdominal condition which involved the peritoneum and caused some pressure on the bile ducts as a second possibility, and not so logical.

Suddenly we have a change in the picture and he complains of very acute abdominal pain. He is brought into the hospital with fairly rapid increase in the ascites, a good deal of distention at any rate, which causes dyspnea. They mention the fact that the left cardiac border is out but not enlarged, simply a displacement. He comes in with definite jaundice. He is a very weak man. No organs were felt so we cannot tell anything further from the physical examination. Inasmuch as he lost a great deal of weight he probably presented the appearance of one who had lost weight from the diaphragm up and not from the diaphragm down. There is no mention of edema.

Acute pain associated with jaundice and ascites can be associated with intrahepatic disease. We have seen a certain number of patients who have had either acute or chronic intrahepatic disease with episodes of jaundice, ascites, and acute belly pain. It can occur with cirrhosis of any type, more frequently with the biliary type; and it can occur with acute yellow atrophy. It is conceivable that in a man coming in practically in shock with some jaundice and with very severe belly pain we would have to consider an acute surgical emergency, such as stone in the common duct or acute pancreatitis. So far as I know these conditions are not associated with ascites, but often with distention. He had localized tenderness. They do not speak of him as being in extreme shock. He has not the slow pulse that goes with pancreatitis. All of these things point against an acute surgical emergency.

If he has cirrhosis with jaundice, ascites, and belly pain, one would expect to get in most instances an anemia. He has a red cell count of 4,700,000, and a hemoglobin of 60 per cent. I

think probably the red blood cell count is not correct, or at least is a false high figure, because he complains in the first place of being very dry, and in the second place it is noted that the tongue is particularly dry; and the red count may be artificially raised by concentration of the blood. He may still have anemia, and, with a hemoglobin of sixty, one might assume that he had anemia for some time but not enough to cause particular symptoms.

The white cell count of 2,500 is very interesting. It seems to me to point to one of two things, either a very acute surgical condition with no localized resistance at all, which is possible; but it seems to me more likely to fit in with the rest of the picture that has gone on for six months and may be chronic intrahepatic disease with leukopenia. It is very often noted in such conditions.

Of course he may have had a hemorrhage from a varix which brings him in in shock, with rapid pulse and apparent dehydration; but he has not vomited blood; and he has watery stools. In the absence of positive findings I should say there is no need for assuming that he had a hemorrhage which might have been the cause of his rapid death.

I cannot believe he has something perforating in the belly, giving abdominal fluid and causing an acute emergency.

I think the logical diagnosis is chronic intrahepatic disease, cause unknown, but probably an infectious type with sudden acute emergency which occasionally occurs in the nature of acute or subacute yellow atrophy. We can rarely get subacute yellow atrophy with no jaundice, or practically none. Any other diagnosis, as cancer, has got to be made on the basis of statistics and I do not see any point in making any other diagnosis. If there is such a thing here, I do not see how one can make the diagnosis. I should think the diagnosis would be cirrhosis of the liver, with acute yellow atrophy.

Dr. Francis T. Hunter: When we first saw this patient he was next door to being moribund. The interesting thing was the acute pain, which I did not realize was a common symptom of chronic intrahepatic disease. The second thing that interested us was the curious area of discoloration around the umbilicus. It suggested that he had hemorrhage which had infiltrated into the tissues in this region. We brought up the question of ruptured viscus and the question of acute abdominal emergency, but after taking everything into consideration we decided that he had an intrahepatic disease. There was no evidence from anything in the family history that he had taken cinchophen.

I should like to answer one question about the laboratory work. The hemoglobin was done by the Tallqvist method and was worthless; so that he might not have had any anemia.

He was discharged with the probable diagnosis of subacute yellow atrophy, and question of cirrhosis and intrahepatic disease of some type related to that group.

Clinical Diagnosis

Portal cirrhosis.

Anatomic Diagnoses

Subacute yellow atrophy of liver.
Ascites.
Peritonitis, generalized acute.
Subphrenic and pelvic abscesses.
Multiple petechial hemorrhages of small bowel, abdominal wall, visceral and parietal pleurae, pericardium, thymus and omentum.
Pleuritis, acute fibrinous, left.
Jaundice, very slight.
Multiple adenomata of adrenals.

Pathologic Discussion

Dr. Tracy B. Mallory: The autopsy showed a very small liver, weighing about 1,000 grams. It was grossly nodular, evidently the end result of an atrophy, but not a very fresh process. The left lobe was almost entirely missing. The remaining liver tissue was made up almost entirely of a slightly hypertrophied grossly nodular right lobe.

The immediate cause of death, and the discoloration around the umbilicus were unusual features. He had an intense purulent peritonitis for which we could find no source whatever. In a good many jaundice cases, and quite regularly in purpura, the presence of an inflammatory lesion determines the localization of the purpuric process. This man's omentum being acutely inflamed from the peritonitis was loaded from end to end with hemorrhagic foci, and the staining around the umbilicus was evidently due to hemorrhage in the parietal peritoneum, the overlying tissues at this point being so thin that the hemorrhage could be seen through the abdominal wall.

A Physician: How much fluid was in the abdomen?

Dr. Mallory: There were several liters. It was not actually measured.

A Student: I should like to inquire whether any relation can be assumed between the accident and the cirrhosis.

Dr. Mallory: I should not think so. One has to assume a chemical rather than mechanical cause.

A Physician: Have you seen atrophied liver with purulent peritonitis before?

Dr. Mallory: I do not remember any other cases of "primary" peritonitis. One does see peritonitis in cirrhosis cases that have been frequently tapped. One is always tempted to assume that the infection is due to the tapping. This case shows that such a conclusion is not always justifiable.

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PSYCHIATRY, PAST AND PRESENT

OF the many subdivisions of medicine, psychiatry is, perhaps, the least stable of the specialties. Based upon no solid rock of ascertained fact, it has suffered much in the past from the ever-shifting sands of conjecture and speculation. Much of it to-day resembles only a museum of models, outmoded by progress and replaced by other models based on further conjecture. Even its disease concepts are vague and uncertain. "Dementia praecox" and "manic-depressive insanity" are largely the results of medical theorizing, without definite boundaries or known etiology. What one considers as dementia praecox to-day, moreover, is not by any means a syndrome that can be defined and many psychiatrists feel that the disease, if one can call it such, is only a phase or a reaction to endocrine imbalance. If so much of psychiatry is on a conjectural basis, is it essential to teach it to the medical student?

This question is considered in a thoughtful paper by Dr. Stearns, dean of the Tufts Medical School, published in this issue.* He points out the psychiatric dilemma and believes that little can be gained by giving more time to the teaching of psychiatry in medical school, if the emphasis continues to be put upon the old-time descriptive method. It seems to matter little whether the student knows all the signs and symptoms of the classic mental diseases. What he should know, however, is the method of a psychological approach to the patient's personality. Psychiatry, which does not even need to be classed as a specialty, should so sensitize "the student's mind that he is always interested in an appraisal of the personality with which he has to deal." This is the duty of the whole medical staff, under the leadership of a psychiatric department.

Perhaps we need less psychiatry taught to our students and more to the faculty! Few members of the teaching staff of our great medical schools realize the value of social and medical psychology. Trained in college by an overdose of mathematics and in the medical school by a heavy schedule of anatomy, the majority of our teachers in medicine, surgery and the other specialties, lack the background for imparting to their students in various departments the psychiatric approach to patients. The wise import of Dr. Stearns' contribution is to keep to a minimum the number of hours given in medical schools to unstable psychiatry, as such, and to increase the time allotted to medical psychology, especially as it can be taught as a part of all the clinical branches of medicine. With such a broad viewpoint, modern medicine should be entirely in accord.

*Page 574.

NEW WORLDS TO CONQUER

MEDICINE, in the era of pathology, has concerned itself to a large degree, and frankly, with the fascinating study of disease. As new methods of analysis were developed—gross pathology, microscopic pathology, physiological chemistry—new vistas were opened up and new avenues were laid out, down which the search for more exact diagnostic procedures and better therapeutic results might lead. Bacteriology, asepsis and anesthesia gave to surgery an impetus which is still carrying it forward in a continuous triumphal march; bacteriology and the intricate complexities of modern chemistry have made our new found medicine seem like a thing of magic.

The span of life has been lengthened as these advances have been made along the skirmish line where the human brain is constantly pitted against the forces of disease. One evidence of this is a shift in the character of the causes of death as more and more individuals survive the mortalities of infancy and the devastating infections of early life, moving into that age group where the wear and tear of existence finally claims its victims through the agencies of the

degenerative diseases. Here is a harder battle to be fought, for so far as our present knowledge goes, each life must eventually come to a close for some adequate reason or another.

What humanity asks for with ever-increasing emphasis, however, and what our profession fails to give any adequate guarantee of, and, indeed, fails to consider in all its seriousness, is the joy of nearly perfect health. Much of the pioneering has been done; existence has been guaranteed to a reasonable extent; we must now turn our attention more thoughtfully to increasing the joy of living. The advertising pages of our popular periodicals show that the need is felt, for where the salesmen of modern business turn their sharp noses there is a scent to be followed.

We must continue and increase our study of disease and maintain such holds as we have on it, but at the same time we must pay more attention to the acquisition and the maintenance of positive health—to the small important factors of body mechanics and hygiene and nutrition, of rest and exercise, of fatigue and endurance, both physical and mental.

The battle against pneumonia must be waged relentlessly, but of equal importance, so far as the economy and the joy of living are concerned, is the common cold, uncomplicated. Tuberculosis of the spine is of less importance to millions than are flat feet. The diseases due to gross vitamin deficiencies are well known; we must consider with even greater seriousness, as they are brought to our attention, the possibilities of minor deviations from health from the same causes, which, though less obvious, may affect millions. Dental caries, in the aggregate, is a more important departure from the normal than is scurvy.

The public is clamoring to know the way to better health. The answer must not be left to the bakers, the mattress makers, the California fruit growers or the manufacturers of any brand of tooth paste.

MEDICAL RELIEF FOR THE INDIGENT OF BOSTON

THE important letter of the Director of the Boston Dispensary appearing on page 603 of this issue should be read by those who are interested in the problems of dealing with illness in Boston. With great concern for those who have been served by the Dispensary, the management is trying to carry on so far as its resources will permit. It has devised plans for doing its best under the circumstances. Where it and other charitable organizations are unable to give the necessary service, measures should be devised for the care of all who need medical attention. This subject is being considered in its broadest aspects by all concerned, and detailed plans will, in all probability, be worked out which will meet existing needs. Boston has always shown its desire to care for her unfortunate people.

TENTH CLINICAL CONGRESS OF THE CONNECTICUT STATE MEDICAL SOCIETY

THIS convocation held in New Haven, September 18, 19, and 20 was a great success. Six hundred and forty physicians of Connecticut and other states registered and attended, an increase of three times the number of the preceding year. The published schedule was followed by dinners and private meetings of the obstetricians and psychiatrists.

The reason for the large attendance was probably that the program was of unusual excellence. Among the important papers Hamman and Martland's discussion of the problems of sudden death, Bayne-Jones on vaccine therapy and Osgood's discussion of arthritis were especially commended.

The success of these meetings has been in large part due to the organizing ability of Dr. Creighton Barker who has been able to secure the coöperation of leading men in the several departments of medicine.

THIS WEEK'S ISSUE

CONTAINS articles by the following named authors:

BACKUS, HAROLD S. M.D. Long Island Medical College 1903. Urologist, Mt. Sinai Hospital, Hartford, Connecticut. Urologist, Hartford Municipal Hospital. Consulting Urologist, Manchester Memorial Hospital, Manchester, Connecticut. His subject is "Traumatic Rupture of Kidney with Symptomatology of Acute Abdomen." Page 563. Address: 99 Pratt Street, Hartford, Connecticut.

HEPBURN, THOMAS N. A.B., A.M., M.D. Johns Hopkins University School of Medicine 1905. F.A.C.S. Urologist, Hartford Hospital. Consulting Urological Surgeon, New Britain General Hospital, Rockville General Hospital, Charlotte Hungerford Hospital, Manchester General Hospital, Meriden General Hospital, and Bristol Hospital. His subject is "Ureterorectal Anastomosis in a Woman with a Single Kidney Because of Tuberculosis of the Bladder." Page 564. Address: 179 Allyn Street, Hartford, Connecticut.

NEUSWANGER, C. H. B.A., M.S., M.D. Harvard University School of Medicine 1923. F.A.C.S. Clinical Instructor in Urology, Yale Medical School. Assistant Attending Surgeon, New Haven Hospital and New Haven Dispen-

sary. Attending Urologist, St. Mary's and Waterbury Hospitals. His subject is "Observations on the Experimental Transplantation of the Ureter." Page 565. Address: 99 North Main Street, Waterbury, Connecticut.

MANWELL, EDWARD J. A.B., M.D. University of Rochester School of Medicine 1930. Instructor in Surgery, Yale University School of Medicine. Resident Surgeon, New Haven Hospital and Dispensary. Former Resident Urologist, New Haven Hospital and Dispensary. His subject is "Urinary Symptoms in Relation to Trichomonas Vaginalis Infestation." Page 567. Address: New Haven Hospital, New Haven, Connecticut.

JENKINS, RALPH H. M.D. Medical College of Virginia 1916. F.A.C.S. Assistant Clinical Professor of Urology, Yale University School of Medicine. Attending Urologist, New Haven Hospital. Address: New Haven Hospital, New Haven, Connecticut. Associated with him are

DEMING, CLYDE L. B.A., M.D. Yale University School of Medicine 1915. F.A.C.S. Clinical Professor of Urology, Yale University School of Medicine. Address: New Haven Hospital, New Haven, Connecticut. And

VAN WAGENEN, GERTRUDE. Ph.D. Address: New Haven Hospital, New Haven, Connecticut. Their subject is "Comparative Histology of the Prostate and Prostatic Urethra in Man, Monkey and Rat from the Viewpoint of the Anatomical Origin of Prostatic Hypertrophy." Page 569.

DR. DEMING also contributes "The Application of Bacteriological, Physiological and Histological Principles to Prostatic Surgery." Page 572.

STEARNS, A. WARREN. M.D. Tufts College Medical School 1910. Dean, Tufts College Medical School. His subject is "The Future of Psychiatry in Medical Schools." Page 574. Address: 416 Huntington Avenue, Boston, Massachusetts.

CAVE, EDWIN FRENCH. A.B., M.D. Harvard University School of Medicine 1924. Orthopedic Surgeon to Out-Patients, Massachusetts General Hospital. Assistant Orthopedic Surgeon, New England Peabody Home for Crippled Children. Assistant in Orthopedic Surgery, Harvard Medical School. His subject is "Tuberculosis of the Spine in Children. Based on a Study of Eighty-Nine Cases from 1920 to 1930 Inclusive." Page 578. Address: 264 Beacon Street, Boston, Massachusetts.

MACDONALD, WILLIAM J. M.D. University of Sydney Medical School 1914. Chief, Department of Dermatology and Syphilology, Carney Hospital. Consultant in Cutaneous Diseases, Cambridge City Hospital. Formerly: Head of Department of Dermatology and Syphilology, 24th Stationary Hospital, Egypt; Graduate Assistant in Department of Syphilis, Massachusetts General Hospital; and Assistant in Department of Syphilis, Massachusetts General Hospital. His subject is "Chemical and Electrogalvanic Burns of Tongue." Page 585. Address: 270 Commonwealth Avenue, Boston, Massachusetts.

EDWARDS, EDWARD A. M.D. Tufts College Medical School 1928. Assistant in Surgery and Member of the Circulatory Clinic, Boston City Hospital. Instructor in Surgery, Tufts College Medical School. Address: 1571 Beacon Street, Brookline, Massachusetts. Associated with him is

BIGURIA, FERNANDO. A.B., B.S., M.D. National University of Guatemala 1929. Research Fellow in Medicine, Tufts College Medical School. Address: 390 Marlborough Street, Boston, Massachusetts. Their subject is "A Comparison of Skiodan and Diodrast as Vasographic Media, With Special Reference to Their Effect on Blood Pressure." Page 589.

REGGIO, A. WILLIAM. A.B., M.D. Harvard University Medical School 1912. F.A.C.S. Instructor in Surgery, Harvard Medical School. Surgeon to Out-Patients, Massachusetts General Hospital. Surgeon to Brookline Board of Health Hospital. Consulting Surgeon, Massachusetts Charitable Eye and Ear Infirmary. His subject is "A Bone Plate Screw Holder." Page 593. Address: 374 Marlborough Street, Boston, Massachusetts.

The Massachusetts Medical Society

STATED MEETING OF THE COUNCIL

A STATED meeting of the Council of the Massachusetts Medical Society will be held in John Ware Hall, Boston Medical Library, 8 The Fenway, Boston, on Wednesday, October 3, 1934 at 12 o'clock, noon.

Business:

1. Call to order at 12, noon.
2. Reading record of last meeting in abstract.
3. Report of Committee of Arrangements for Annual Meeting.
4. Report of Committee on Membership and Finance.
5. Reports of committees to consider petitions for restoration to the privileges of fellowship and appointment of new committees.
6. Report of committee appointed to consider the proposed resolution of the Legislative Committee of the Hampden District Medical Society regarding House Bill No. 118.

7. Appointment of an auditing committee.
8. Fill any vacancies in the offices of the Society.
9. *Incidental Business.*

WALTER L. BURRAGE, *Secretary.*

Brookline, September 26, 1934.

Councilors are reminded to sign one of the attendance books before the meeting.

SECOND ANNUAL POSTGRADUATE MEDICAL EXTENSION COURSE

GIVEN BY THE MASSACHUSETTS MEDICAL SOCIETY
FALL AND SPRING, 1934-1935

INFORMATION

Time of Course

First week of October to mid-December. Mid-March to June. No sessions during Boston meeting of American College of Surgeons, October 15 to 19.

Place

Your own district.

Number of Exercises

A course will consist of ten weekly "sessions"; subjects listed in groups of three sessions each, except one elective. Choose three subjects and one elective.

Duration of Each Session

Two hours.

Instructors

Instructors will go in teams of two.

Subjects Actually Given

After you mail your choices the votes are counted; majority vote is the rule. For details see your district chairman.

Registration

A fee of five dollars for course of ten sessions.

CURRICULUM

Choose three groups listed 1, 2, 3, etc. Each group requires three sessions. Choose one elective, making a total of ten sessions.

1. *Cardiovascular Disease.*

Session 1. *Diagnostic Methods.*
History and Physical Examination.
X-ray.
Basal Metabolism.
Vital Capacity.
Electrocardiogram.

Session 2. *Types of Heart Disease and Their Pathological Manifestations.*
Rheumatic Heart Disease and Damaged Valves.
Syphilitic Aortitis and Its Effect on the Heart.
Hypertension.
Coronary Disease.
Other Etiological Factors.

Session 3. *Disorders of Function of the Heart and Their Treatment.*
Congestive Failure.
Angina Pectoris.
Arrhythmias.

This important subject is repeated in more detail this year; 100 per cent request last year shows the interest of the profession in this most common cause of death.

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2. *Endocrinology.*

Session 1. *General Principles of Treatment.*
Uniglandular, Polyglandular Therapy, Hypodermic vs. Oral Dosage.

Session 2. *Diseases and Disturbances of the Thyroid and Parathyroid Glands.*
Thyroid Hypofunction.
Differential Diagnosis.
Effect on Special Functions—Circulation, Renal Function, Sex Development and Function—Importance in Sterility and Miscarriage—Nervous Disturbances.
Thyrotoxicosis—Differential Diagnosis.
Psychic Disturbances, Effect on Sex Functions, Menstruation, Fertility.
Parathyroid Tumor—Diagnosis and Treatment.

Session 3. *Diseases and Functional Disturbances of Pituitary Gonad Group.*
Physiology; Pathology.
Effect on Growth, Maturity, and Fertility.
Normal Menstruation and Functional Disturbances — Menorrhagia, Amenorrhea, Oligomenorrhea.
Treatment: Use of Pituitary Extracts. Use of "Female Sex Hormones."

The importance of this subject is appreciated by all doctors; recent advances in diagnosis and treatment are enlightening.

---

3. *Industrial Medicine.*

Session 1. *Lung Disease as an Occupational Hazard.*
Silicosis and Other Chest Lesions.
Lead Poisoning.

Session 2. *Occupational Dermatitis,* and Other Skin Lesions Commonly Treated in General Practice.
Relations of Industrial Physician to Private Doctor.

Session 3. *Industrial Surgery and Medico-Legal Problems.*
Modern Treatment of Burns.
Minor Surgical Problems.
Back Strain and Joint Injuries.
The Physician's Medico-Legal Problems in Industrial and Automobile Cases.

Workmen's Compensation and Compulsory Automobile insurance laws have made this subject of direct interest to practically every Massachusetts M.D. Over one hundred and seventy thousand industrial cases in 1930 involving payments of slightly less than ten million dollars in fees was the peak; in 1933 ninety-six thousand cases, fees paid, five million eight hundred thousand dollars.

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4. *Nutrition in Health and Disease.*

Session 1. *Review of the Principles of Dietetics.*
Carbohydrate Proteins and Fat.
Metabolism. Water Balance in the Body.
Treatment of Obesity and Malnutrition.

Session 2. *The Dietetic Treatment of Deficiency States.*

Anemias. Minerals in the Diet.
Avitaminosis, etc.

Session 3. *The Dietetic Treatment of:*

Diabetes — Constipation
Peptic Ulcer — Diarrhea

An accurate working knowledge of these subjects will improve any doctor's practice.

---

5. *Obstetrics and Gynecology.*

Session 1. *Pregnancy and Labor.*

Aschheim-Zondek and Similar Reactions.
Present-day Pelvimetry.
Deficiency Diseases and Blood Changes. Diet.
Roentgenology in Obstetrics.
Management of Heart Cases and the Toxemias.
Amnesia, Methods, Technique.
The Oxytocics.
Artificial Induction of Labor.
Episiotomy and "Perineal Forceps."

Session 2. *Pathology of Labor.*

Delay, Inertia, Disproportion, Faulty Mechanism.
Management of the Bleeding Cases.
Delivery Operations.
Emergencies of the New-Born.

Session 3. *Gynecology.*

Pelvic Inflammation—Diagnosis and Treatment.
Gonorrheal. Tuberculous.
Post-Partal and Post-Abortal.
Other Forms.
Endocervicitis and Vaginitis.
Office Procedures in Examination.
Office Treatment.
Precancerous Lesions and Cancer of the Uterus.
Diagnosis. Endoscopy. Biopsy.
Treatment by Radium or X-Ray.
Surgery.

Modern methods of diagnosis and treatment are taught in this series. Time devoted to this course will be well repaid.

---

6. *Pediatrics.*

Session 1. *Diseases of Upper Respiratory Tract and Complications.*

Session 2. *Physical Examination.*

Infants and Children.
Differential Diagnosis.

Session 3. *Principles of Nutrition of Infants and Children.*

Practical Application of Principles.

The proper treatment of "Just a Cold" is an art as well as a science. This is a course for everyday practical use.

---

7. *Surgery.*

Session 1. *Minor Surgical Problems.*

Furuncle and Carbuncle—Cervical Abscess.
Infections of Fingers and Hand, Toes and Foot.
Treatment of Dirty Wounds—Question of Antitetanic Serum.
Hemorrhoids; Fissure and Fistula-in-Ano. Pruritus Ani.

Minor Orthopedic Lesions.

Foot Strain—Faulty Arches.
Sprains of Ankle, Knee, Wrist.
Dislocated Semilunar Cartilage.
Acute and Chronic Backstrain and Other Types of Backache.
Painful Shoulders. Hallux Valgus.
Athletic Injuries, Muscle Bruises, etc.

Session 2. *The Acute Abdomen.*

Diagnosis from Type and Location of Pain, Constitutional Signs and Symptoms.
Differentiation from Medical Lesions, Pneumonia, etc.
Acute Pain in the Epigastrium and the Four Quadrants.
Intestinal Obstruction in Large and Small Bowel, Acute and Chronic.
Intra-Abdominal Trauma, Perforation and Hemorrhage.
Hemorrhage Into and From the Gastro-Intestinal Tract.
Ulcer of Stomach and Duodenum.
Meckel's Diverticulum.
Colitis, Tuberculosis, Polyps, and Cancer of the Colon.

Session 3. *Symposium on Common Fractures.*

Rib. Colles'. Pott's. Both Bones Leg. Arm, Upper and Lower. Fingers and Toes.

Physical Therapy in Office and Home Practice.

Here are important and demanding problems. Best methods of diagnosis and treatment will be presented.

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8. *Elective Session No.* 10. (Choose One Number.)

1. *Pulmonary Tuberculosis.*

Early Diagnosis.
The Use of X-ray in Combination with Physical Signs.

2. *Amebiasis and Parasite Diseases Common in New England.*

3. *The Common Neuroses and Their Treatment in Private Practice. The Psychoses—Early Diagnosis.*

4. *Laboratory Procedures Useful in Office Practice and Home Treatment.*

Guides to References; Textbooks and Journals.
Medical Economics.

5. *Dermatology and Syphilis.*

Ten Common Skin Diseases—Diagnosis and Treatment.
Syphilis, Modern Treatment. The Use of Neosalvarsan, Tryparsamid, Bismuth, Mercury, Potassium Iodide, etc., in Office Practice.

6. Any Subject in Groups not Already Chosen.

7. Your Own Choice of Subject.

Wide latitude of interest makes a more intelligent profession.

DISTRICT CHAIRMEN OF POSTGRADUATE INSTRUCTION

Barnstable
John I. B. Vail, Hyannis.

Berkshire
Albert C. England, Pittsfield.
George S. Reynolds, Pittsfield.

Bristol North
Arthur R. Crandell, Taunton.
William M. Stobbs, Attleborough.

Bristol South
Harold E. Perry, New Bedford.

Essex North
Francis W. Anthony, Haverhill.

Essex South
Walter G. Phippen, Salem.

Franklin
Halbert G. Stetson, Greenfield.

Hampden
George L. Schadt, Springfield.

Hampshire
Robert B. Brigham, Northampton.

Middlesex East
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Middlesex North
Frederick P. Murphy, Lowell.

Middlesex South
Charles E. Mongan, Somerville.

Norfolk
Hugo B. C. Riemer, Norwood.

Norfolk South
David L. Belding, Hingham.

Plymouth
Walter H. Pulsifer, Whitman.

Suffolk
Reginald Fitz, Boston.

Worcester
Erwin C. Miller, Worcester.

Worcester North
Edward A. Adams, Fitchburg.

If any doctors have not received a program, kindly notify the Secretary, 8 Fenway, or see the district chairman.

EXECUTIVE COMMITTEE
DR. REGINALD FITZ, *Chairman*,
DR. ROBERT B. GREENOUGH,
DR. JOSEPH W. O'CONNOR,
DR. LEROY E. PARKINS, *Secretary*.

MISCELLANY

THE OPENING EXERCISES OF THE BOSTON UNIVERSITY SCHOOL OF MEDICINE

On September 20, 1934, the opening exercises of the Sixty-Second Annual Session of the Boston University School of Medicine were held in the Evans Auditorium.

Dr. A. S. Begg, Dean of the Medical School, called the meeting to order at eleven o'clock, and after an invocation by Professor Clyde E. Wildman, Southworth Professor of Old Testament History and Religion, introduced Dr. Daniel L. Marsh, President of Boston University. Dr. Marsh found in his experiences during a visit to Interlaken, when impressed with the beauty and majesty of the Jungfrau, that his impulse to ascend the mountain led him to expect that that accomplishment would be the acme of his ambition. Having fulfilled his dreams on reaching the heights of this mountain, his vision was so expanded that he realized the immensity of opportunities for greater adventures. Applying the reactions to this experience in Switzerland to the assembled students, he wished all to realize that although graduation from the school seems to be the great objective, when this has been accomplished, broader and more satisfying experiences lie ahead in the services to be rendered and the intellectual development incident to the physician's life if one takes advantage of its opportunities.

He assured the student body that the medical school opened the way to the highest achievements if one has the ambition to profit by the opportunities provided by the school.

Dr. Marsh emphasized the benefits which will come to those from other preparatory schools through participation in the social and intellectual activities of the University. He gave cordial assurance of the interest of Dean Begg, and all members of the faculty in the welfare of the students.

Dean Begg followed Dr. Marsh in addresses to the several classes explaining the work which has been planned for the 1934-1935 year.

One interesting feature of the exercise was the introduction to the students of two benefactors of the school, Dr. J. Emmons Briggs, Professor Emeritus of Surgery, long on the faculty of Boston University School of Medicine, and Mr. Brewer of the Brewer Pharmaceutical Company of Worcester.

In approaching an invitation to the students to join the Glee Club, Mr. James Houghton, Assistant Professor of Church Music and Worship in the School of Theology of Boston University, gave a short and humorous reference to the history of glee clubs, and then sang, "Hail, Boston University," the words of which were written by President Marsh. This was enthusiastically applauded.

After the close of the formal exercises, a luncheon was served to the faculty and guests, and an opportunity given to inspect the changes which have been made in the buildings and accommodations, in order to enable the faculty to work to better advantage.

CURTAILMENT OF HOME MEDICAL SERVICE

The Boston Dispensary
25 Bennet Street
Boston, Massachusetts
September 20, 1934.

We regret to inform the social and health agencies of Boston that, because of lack of financial support, the Boston Dispensary is forced to make drastic curtailments in its visiting doctors' service to the sick poor in their homes.

In the application of this curtailment, we are confronted with the necessity of determining whether

our policy shall be one of geographical reduction or of reduction for some other, possibly economic, reason; in other words, whether we shall (1) drop some districts entirely, withdrawing service from all persons in those districts dropped, or (2) continue work in all districts, withdrawing service from some individuals for other clearly defined reasons.

Analysis reveals that many otherwise self-supporting families, not on public relief, are unable to provide for medical emergencies. For these, the only resource would be public medical aid, if private medical aid were to be withdrawn. Other families in which medical emergencies arise are already dependent upon public relief, including eligibility to public medical relief.

It has seemed most logical to us to use such funds as we are able to secure for the benefit of those not receiving public relief (for whom there are no other resources), since the other group may look to public funds for medical relief. For this reason, the service will be *withdrawn* on October 1, 1934, from:

(1) Individuals or families, residents of Boston, receiving aid from the Overseers of the Public Welfare;
(2) Mothers' Aid cases;
(3) Individuals receiving Old Age Assistance;
(4) Soldiers' Relief cases.

The service will be *continued*, as heretofore, to persons not receiving City Aid, who are clients of social agencies, the Community Health Association, and to other individuals not able to travel to a clinic, or to employ a private physician.

In order to avoid unnecessary delay in getting the call to its proper destination, persons referring calls are requested first to ascertain whether the family or individual is a proper case for acceptance under the above policy. Persons not admissible to the Dispensary's service should be advised to apply to the district agent or visitor responsible for social handling of relief for the above-mentioned groups.

FRANK E. WING, *Director.*

CORRESPONDENCE

AN APPEAL TO THE HOSPITALS OF BOSTON

September 19, 1934.

Editor, The *New England Journal of Medicine*,
Sir:

I herewith submit a communication which has been presented to the Trustees of the Boston City Hospital which will in turn be presented to other hospitals in Boston.

I am sure that it would interest your readers to have the aforesaid communication published in the *Journal*.

Yours very truly,
CHARLES MALONE, M.D.,
Secretary of Committee.

45 St. John Street,
Jamaica Plain, Mass.

To the Trustees Boston City Hospital:

A Committee has been appointed by the Norfolk District Medical Society (a component part of the Massachusetts Medical Society) to devise means to end the extraordinary abuse of Medical Charity in Boston, which abuse is having a demoralizing effect on the Medical Profession.

The Committee, therefore, ask the Trustees of the Boston City Hospital for the following changes in the mode of admission to the O. P. Department and the Hospital Proper:

(1) That patients coming into the O. P. Department for the first time be treated, that they then be given a card and told that in order to get further treatment they must have this card endorsed by a physician or the Welfare Department (a sample card is herewith recommended).

(2) That admission to the Hospital Proper be made only on the recommendation of physicians, the O. P. Department, or the Board of Trustees.

(3) That ambulance calls be made only on the request of Physicians, Police Officers, Board of Trustees, or in any Emergency.

The Massachusetts Medical Society at its annual meeting in Worcester, June, 1934, endorsed in principle the foregoing requests.

(Signed) CHARLES MALONE, M.D., *Secretary.*

RECENT DEATHS

CONWAY — FRANCIS BERNARD CONWAY, M.D., of 229 Harrison Avenue, Boston, died of coronary thrombosis at the Boston City Hospital, September 17, 1934.

He was born in 1869, and graduated from the Tufts College Medical School in 1903, and had practiced in Boston for thirty-one years.

He joined the Massachusetts Medical Society in 1922. He was a life member of the Elks.

HAMILTON—ANNIE LEE HAMILTON, M.D., of Sandwich, Massachusetts, died at the Glenside Hospital, Jamaica Plain, September 23, 1934.

Dr. Hamilton was born in Nova Scotia in 1864 and trained as a nurse at the Massachusetts General Hospital, and later graduated in medicine from the Woman's Medical College of the New York Infirmary for Women and Children in 1898. She served as house officer of the New England Hospital. She joined the Massachusetts Medical Society in 1899 and retired in 1930. For several years she had an office in Copley Square and was consulting physician to the firm of W. F. Schrafft and Sons, Corp. She is survived by a sister and a brother.

NOTICES

CLINIC AT THE PETER BENT BRIGHAM HOSPITAL

At 3:30 P.M. on Thursday, October 4, in the amphitheater of the Peter Bent Brigham Hospital

Dr. Henry A. Christian, Physician-in-Chief, Hersey Professor of the Theory and Practice of Physic in the Harvard Medical School, will give a medical clinic. To it are cordially invited practitioners and medical students. These clinics will be repeated on Thursdays, October to May.

On Saturdays in the wards of the Peter Bent Brigham Hospital, from 10 to 12, staff rounds will be conducted by Dr. Christian. These are open to all physicians.

LAWRENCE CANCER CLINIC

Lawrence, Mass.,
September 19, 1934.

To the Physicians of the North Half of Essex County:

Dear Doctor:

The regular Lawrence Cancer Clinic, to be held at Lawrence General Hospital, 1 Garden Street, Lawrence, upon Tuesday, October 2, at 10:00 A.M., will be a Demonstration Clinic with Channing C. Simmons, M.D., of Boston, Associate in Surgery at Harvard University Medical School, acting Surgeon-in-Chief to Collis P. Huntington Memorial Hospital, and member of the Cancer Commission of Harvard University, Boston, present as consultant. You are invited to accompany any of your patients whom you desire shall have this service, or to send them with a note, and a report will be returned to you. This service is gratis. Your attendance at the Clinic is always welcome.

This Clinic is endorsed by the Committee on Postgraduate Instruction of the Massachusetts Medical Society.

Committee
ROY V. BAKETEL, M.D.,
CHARLES J. BURGESS, M.D.,
FREDERICK D. MCALLISTER, M.D.,
JOHN J. MCARDLE, M.D.,
HARRY H. NEVERS, M.D.,
THOMAS V. UNIAC, M.D.,
J. FORREST BURNHAM, M.D., *Chairman.*

REMOVAL

THOMAS H. LANMAN, M.D., announces the removal of his office to the Children's Hospital, 300 Longwood Avenue, Boston.

REPORT AND NOTICES OF MEETINGS

THE FRANKLIN DISTRICT MEDICAL SOCIETY

The September meeting of the Franklin District Medical Society was held at the Weldon Hotel in Greenfield on September 11, at 11:00 A.M.

The proposed postgraduate course for the coming year was discussed. The following were appointed as members of the program committee for the year: Drs. H. N. Howe, J. E. Moran and C. V. Perry.

Dr. E. Granville Crabtree, of Boston, was introduced by President Marble, and presented for his subject "The Urinary Diseases of Pregnancy." Dr. Crabtree emphasized the importance and frequency of this disease in pregnant women and explained the necessity for proper treatment. The address was illustrated by lantern slides.

FAULKNER HOSPITAL CLINICAL MEETING

The Faulkner Hospital throughout the coming winter months will hold a meeting for its staff and any members of the medical profession who are interested on the first Thursday of each month at 5:00 P.M. at the hospital. These meetings will last approximately one hour. Half of the time will be given to a clinical pathological discussion of the interesting cases which have come to autopsy during the preceding month and there will be a short talk on some topic of particular interest by some one especially qualified to talk on the subject.

The first meeting will be held on Thursday afternoon, October 4, at 5:00 P.M. After the clinical pathological conference Dr. Arthur A. Cushing will discuss some interesting cases of agranulocytosis which have recently been observed at the hospital. All members of the profession are cordially invited.

ESSEX NORTH DISTRICT MEDICAL SOCIETY

The ninety-third quarterly meeting will be held at Raymonds Sunnyridge Farm, Amesbury Road, Amesbury, on Wednesday, October 17, 1934, at 12:30 P.M. Dr. William F. MacKnight and associates will discuss the interesting adjustments of medical practice to local economic conditions under the Jackson Administration.

The semi-annual meeting will be held in January and the annual meeting in May, time, place and subject to be announced.

E. S. BAGNALL, M.D., *Secretary.*

NEW ENGLAND PEDIATRIC SOCIETY

Combined meeting of the American Academy of Pediatrics (Region 1), New England Pediatric Society, Philadelphia Pediatric Society, and the New York Academy of Medicine Pediatric Section.

Two sessions on Friday, October 19, at 9:30 A.M. and 2:30 P.M.

One session on Saturday morning, October 20, at 9 A. M.

Dinner Friday evening, October 19, at the Hotel Ambassador.

Headquarters—Hotel Ambassador—Park Avenue and 51st Street, New York City.

GERALD HOEFFEL, M.D., *Secretary-Treasurer.*

ACADEMY OF PHYSICAL MEDICINE

ANNUAL MEETING

The Annual Meeting will be held at the Hotel Pennsylvania, New York, October 30 and 31. For information address Arthur H. Ring, M.D., Secretary-Treasurer, Arlington Heights, Mass.

SOCIETY MEETINGS, CONGRESSES AND CONFERENCES

September 28-29—New England Surgical Society will meet at Burlington, Vermont. See page 519, issue of September 13.

October—International Medical Postgraduate Courses. See page 335, issue of August 16.

October 2—Lawrence Cancer Clinic. See page 605.

October 4—Clinic at the Peter Bent Brigham Hospital. See page 604.

October 4—Faulkner Hospital Clinical Meeting. See page 605.

October 7-20—Sixth Annual Medico-Military Symposium, at the Mayo Clinic. See page 382, issue of August 23.

October 8, 9 and 10—Association of Military Surgeons of the United States. See page 473, issue of September 6.

October 9 - April 9—Massachusetts Dietetic Association.

October 9—Tuesday, 8 P.M. "Food Travelogue," Miss Edith Barber, Writer and Consultant, New York City.

November 7—Wednesday, 8 P.M. Forsyth Dental Infirmary for Children, 140 Fenway, Boston, "The Arrest and Control of Dental Caries," Dr. C. L. Drain, College of Dentistry, State University of Iowa.

November 20—Tuesday, 2 P.M. Field Trip: Visit Kitchens of the Hotel Statler.

December 11—Tuesday, 8 P.M. "Food Allergy," Dr. Frank N. Allan, Department of Medicine, Lahey Clinic.

January 8—Tuesday, 8 P.M. "Careers and Natural Abilities," Professor Johnson O'Connor, Human Engineering Laboratory, Stevens Institute of Technology, Hoboken, N. J.

February 12—Tuesday, 8 P.M. "Diabetic Children," Dr. Priscilla White, Joslin Diabetic Unit.

March 12—Tuesday, 8 P.M. "The Effect of Diet on Anemia," Dr. Lewis Diamond, Instructor in Medicine, Harvard University Medical School, Associate Physician, Children's Hospital.

March 19—Tuesday, 2 P.M. Field Trip: Visit Storehouse, First National Stores.

April 9—Tuesday, 8 P.M. "Small Hospital Problems," Miss Margaret Copeland, Superintendent, Free Hospital for Women.

October 15-19—Sixth Annual Session of the Association of Record Librarians of North America at Hotel Kenmore, Boston. Chairman of Committee on Arrangements—Miss Edith M. Robbins, Peter Bent Brigham Hospital, Boston.

October 15-19—American College of Surgeons at Boston. See page 424, issue of August 30, and page 559, issue of September 20.

October 15-19—Thirteenth Annual Congress of Anesthetists. See page 515, issue of September 13.

October 17—William Harvey Society. Speaker: Dr. Emil Novak of Baltimore.

October 19-20—New England Pediatric Society. See page 605.

October 22 - November 2—1934 Graduate Fortnight of the New York Academy of Medicine. See page 1240, issue of June 7

October 30-31—Academy of Physical Medicine. See notice above.

October 31 - November 2—Massachusetts State Nurses' Association, Hotel Statler, Boston. For information write Miss Helene G. Lee, R.N., 420 Boylston Street, Boston.

November 5-9—International Assembly of the Inter-State Postgraduate Medical Association of North America. See page 520, issue of September 13.

December 3-7—The Radiological Society of North America. See notice on page 562, issue of September 20.

April 29 - May 3, 1935—The American College of Physicians will meet in Philadelphia. For information address Mr. E. R. Loveland, Executive Secretary, 133-135 South 36th Street, Philadelphia, Pa.

June, 1935—Medical Library Association will meet in Rochester, N. Y. For details address the Secretary: Miss Frances N. A. Whitman, Librarian, Harvard University Schools of Medicine and Public Health, Boston, Mass.

DISTRICT MEDICAL SOCIETIES

ESSEX NORTH DISTRICT MEDICAL SOCIETY

October 17—See page 605.

FRANKLIN DISTRICT MEDICAL SOCIETY

Meetings will be held on the second Tuesday of November, January, March, and May at the Weldon Hotel, Greenfield, Mass.

CHARLES MOLINE, M.D., Secretary.
Sunderland.

MIDDLESEX EAST DISTRICT MEDICAL SOCIETY

October 10—Woburn.
November 14—Reading.
January 9, 1935—Melrose.
March 13, 1935—Wakefield.
May 8, 1935—Winchester.

K. L. MACLACHLAN, M.D., Secretary.
1 Bellevue Street, Melrose.

WORCESTER DISTRICT MEDICAL SOCIETY

October 10—Wednesday evening. Rutland State Sanatorium, Rutland, Mass. 6:30 P.M.: Dinner. 7:30 P.M.: Scientific program and business session. Announcement of subjects and speakers to be presented at a later date. Dinner complimentary by the Hospital.

November 14—Wednesday evening. Grafton State Hospital, North Grafton, Mass. 6:30 P.M.: Buffet supper. 7:30 P.M.: Scientific program and business session. Announcement of subjects and speakers to be presented at a later date. Buffet supper complimentary by the Hospital.

December 12—Wednesday evening. St. Vincent Hospital, Worcester, Mass. 6:30 P.M.: Dinner. 7:30 P.M.: Scientific program and business session. Announcement of subjects and speakers to be presented at a later date. Dinner complimentary by the Hospital.

January 9, 1935—Wednesday evening. Worcester City Hospital, Worcester, Mass. 6:30 P.M.: Buffet supper. 7:30 P.M.: Scientific program and business session. Announcement of subjects and speakers to be presented at a later date. Buffet supper complimentary by the Hospital.

February 13, 1935—Wednesday evening. Worcester State Hospital, Worcester, Mass. 6:30 P.M.: Dinner. 7:30 P.M.: Scientific program and business session. Announcement of subjects and speakers to be presented at a later date. Dinner complimentary by the Hospital.

March 13, 1935—Wednesday evening. The Memorial Hospital, Worcester, Mass. 6:30 P.M.: Buffet supper. 7:30 P.M.: Scientific program and business session. Announcement of subjects and speakers to be presented at a later date. Buffet supper complimentary by the Hospital.

April 10, 1935—Wednesday evening. Worcester Hahnemann Hospital, Worcester, Mass. 6:30 P.M.: Dinner. 7:30 P.M.: Scientific program and business session. Announcement of subjects and speakers to be presented at a later date. Dinner complimentary by the Hospital.

May 8, 1935—Wednesday afternoon and evening. Annual Meeting of the Worcester District Medical Society. The time and place of this meeting will be announced later.

ERWIN C. MILLER, M.D., Secretary.
27 Elm Street, Worcester.

BOOKS RECEIVED FOR REVIEW

The Autonomic Nervous System. By Albert Kuntz. Second Edition. 697 pp. Philadelphia and London: Lea and Febiger. $7.50.

Modern Treatment in General Practice. Edited by Cecil P. G. Wakeley. 426 pp. Baltimore: William Wood and Company. $4.00.

BOOK REVIEW

Thérapeutique Hydro-Climatique des Maladies non Tuberculeuses de l'Appareil Respiratoire. Par M. Piery. 155 pp. Paris: Masson et Cie, 1934. 20 fr.

A monograph of 165 pages dealing with the effect of mineral waters and spas on the evolution of non-tuberculous affections of the respiratory tract.